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Depressed Mood After Stroke

A Community Study of its Frequency

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A community study on stroke registered 976 patients. Over 60% of survivors at three weeks, six months and 12 months after the stroke were assessed for depressed mood, using the Wakefield self-assessment depression inventory, and on other functional and social activities. At each point, 25–30% of those assessed were depressed; over 50% of patients depressed at three weeks remained so at one year. Factors associated with depression included loss of functional independence, a low level of other activities, a low reasoning ability, and being female and living with someone; path analysis showed that most depression was not explained by these factors. Few depressed patients at six months were on antidepressant medication.

The physical disability caused by stroke is obvious but, despite the attention drawn to the "hidden barriers to recovery" (Adams, 1974), little attention has been paid to the emotional aspects of stroke. Reasons may include the difficulty in assessing emotion accurately, and the feeling that it is only to be expected that stroke patients will be depressed. It is only in the past few years that the frequency of depression after stroke has been investigated. One group have found depression to be present in about 30% of patients (Robinson & Price, 1982; Robinson *et al*, 1983); they have suggested that it may be specifically associated with localised damage near the left frontal lobe (Robinson *et al*, 1984*a*) and that treatment with nortriptyline may relieve it (Lipsey & Robinson, 1984). A second group also found depression in about 30% of patients, and related this to their loss of social activities (Feibel & Springer, 1982).

The pioneering work of these two groups of investigators needs to be replicated. We have investigated the frequency of depressed affect after stroke in a community survey. In this paper we examine the following questions:

(a) How prevalent is depression at various times after stroke?
(b) What is the natural history of depression after stroke?
(c) What factors relate to the presence of depression at any time?

Method

Over 28 months, a total of 976 acute cases were registered in a community survey of stroke-related disability, based on a population of 215 000 patients registered with 96 general practitioners in Bristol. Of these, 249 patients (26%) were not admitted to hospital over the first six months after the stroke. Patients were identified by direct notification from anyone involved in stroke care (e.g. family or hospital doctors, nurses, therapists) and also by obtaining the records of all patients known to have been discharged from any hospital in the county of Avon with a primary or secondary diagnosis of stroke (ICD-9 classifications 430–438; World Health Organisation, 1978). A clinical diagnosis was used for this study, based upon the WHO definition (Aho *et al*, 1980): "Rapidly developed clinical signs of focal (or global) disturbance of cerebral function, lasting more than 24 hours or leading to death, with no apparent cause other than of vascular origin". The diagnosis was confirmed by a neurologist, by personal examination in 713 (73%) cases or by reading hospital notes if a patient had died or was registered late.

Each patient was seen as soon as possible after the stroke, at 3 weeks, at 6–7 months, and at 12–13 months; at each point, information was recorded on many aspects of the neurological, functional, emotional, and social status.

Depression was assessed using the Wakefield self-assessment depression inventory (Snaith *et al*, 1971), which has 12 questions giving a score between 0 and 36; although not validated in the elderly or in stroke, it was derived from the Zung scale, which has been validated in stroke research (Robinson & Price, 1982). In the original validation, a score of 15 or more was found to be the best cut-off to identify those with depression. We have classified those scoring 15–18 as exhibiting 'probable depression' and 19–36 as 'definitely depressed', because there is some evidence that older people tend to have higher scores (Snaith *et al*, 1971). The Hopelessness scale (Beck *et al*, 1974), comprising 20 statements for the patient to agree or disagree with, was also used, but many found it difficult to understand, and so the results were only used to help validate the Wakefield scale. Since the Wakefield inventory was not developed as an accurate diagnostic instrument, but rather as a way of assessing the severity of depression, our results concern the

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TABLE I
Characteristics of 976 patients studied

	Time		
	3 weeks	*6 months*	*12 months*
Died: *n*	350	432	484
Notified late/lost to follow-up: *n*	57	69	58
Aphasic or confused: *n*	190	98	86
Assessed for depression: *n*	379	377	348
Average age: years			
Mean	69.9	69.7	69.3
s.d.	9.6	9.8	10.3
Male: *n*	203 (54%)	209 (55%)	180 (52%)
Living alone: *n*	87 (23%)	88 (23%)	92 (26%)
Not admitted in 6 months after stroke: *n*	159 (42%)	165 (44%)	151 (43%)
Side of weakness: *n*			
Right	170 (45%)	174 (46%)	160 (46%)
Left	178 (47%)	166 (44%)	152 (44%)
Both or neither	31 (8%)	37 (10%)	36 (10%)
Disability–Barthel scores: *n*			
0–14 (severe/moderate)	99 (26%)	48 (13%)	45 (13%)
15–19 (mild)	123 (32%)	128 (34%)	117 (34%)
20 (no disability)	157 (42%)	201 (53%)	186 (53%)
Depression–Wakefield scores			
0–14 (not depressed)	252 (67%)	257 (68%)	240 (69%)
15–18 (probably depressed)	43 (11%)	46 (12%)	45 (13%)
19–36 (depressed)	84 (22%)	74 (20%)	63 (18%)

extent of any feeling of depression or sadness, rather than clinically defined 'depression'.

Social function was assessed using the Frenchay Activities Index (FAI) (Holbrook & Skilbeck, 1983); this records the frequency with which a patient has performed various activities over the preceding months, and gives a score ranging from 0 (no participation) to 45 (full participation in all 15 items). Physical function was measured using the Barthel Activities of Daily Living (ADL) scale (Granger *et al*, 1979). This scale measures ten self-care items, and gives a score of 0–20. A score of 0–9 indicates 'severe' disability; 10–14, moderate disability; 15–19, 'mild' disability; and 20, functional independence. Each patient's reasoning ability (IQ) after the stroke was measured using Raven's coloured progressive matrices (Raven, 1965): this is a non-verbal measure with normative data for the elderly. Motor loss was assessed using the Motricity index (Demeurisse *et al*, 1980), which gives a score ranging from 0 (complete paralysis) to 100 (normal strength). Two self-assessment procedures were also used. Firstly using a visual analogue scale 150 mm long, patients were asked to indicate the degree of recovery of independence they had achieved: the end-points were "the worst you were with your stroke" and "as you were before your stroke". Secondly, using a five-point scale, patients were asked to indicate their depression relative to before their stroke as much less, less, the same, more, or much more (scored 1–5).

Results

Details on the patients seen and time-point assessed are shown in Table I. Patients with a wide range of disabilities and ages were included. The number of patients not assessed due to late notification, and because of communication problems (either aphasia or confusion) is also given. Only ten patients refused to answer the questions. The high proportion of patients not admitted reflects the fact that many of those admitted were severely affected, and died within a short time. The prevalence of a depressed affect at three weeks, six months and one year is also shown: approximately 20% of patients were 'definitely depressed' at any point, and a further 10% 'probably depressed'. The Hopelessness scale was completed by 317 patients at one year: their average score was 5.64 (s.d. 4.02), ranging from 0 to 19, with only 26 scoring 13 or more.

Recovery from depression did occur. Of the 127 scoring 15 or more on the Wakefield scale at three weeks, 26 had died by six months, and a total of 46 had died by one year. At six months, 33 (33%) of the surviving 101 patients scored 14 or less. At one year, 28 (35%) of the surviving 81 patients were not depressed.

To determine how much variability occurred with time, details of the 255 patients with assessments at all three points were analysed: 175 (69%) scored 0–14 at three weeks and 80 (31%) scored more than 15; 133 (52%) were consistently

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TABLE II
Some correlation coefficients with Wakefield scores (all significant at $P<0.01$)

	n	*Coefficient*
At 3 weeks		
Barthel ADL score	379	−0.329
IQ (Raven's matrices)	331	−0.257
Motricity score	342	−0.324
At 6 months		
Barthel ADL score	377	−0.219
IQ (Raven's matrices)	345	−0.146
Current social activities (FAI score)	375	−0.353
Loss of social activities (FAI score)	357	+0.229
Carer's depression (Wakefield)	192	+0.345
Self-image of recovery (line scale)	293	−0.430
Self-image of mood change	359	+0.539
At 1 year		
Barthel ADL score	348	−0.247
IQ (Raven's matrices)	330	−0.203
Current social activities (FAI score)	348	−0.371
Carer's depression (Wakefield)	200	+0.253
Other correlations		
Wakefield 3 week and 6 month scores	312	+0.690
Wakefield 3 week and 1 year scores	261	+0.630
Wakefield 6 month and 1 year scores	299	+0.750
Wakefield 6 month and Barthel 3 week scores	365	−0.165

'not depressed', while 44 (17%) were consistently 'depressed' at all three points. Thirty-nine (15%) depressed patients had recovered by one year, but 14 (5%) who were not initially depressed became so by six months, while 25 (10%) became depressed by one year; 19 (7%) patients fluctuated between the three assessment points.

One can conclude that approximately half of the patients who were depressed at one year had been depressed since soon after their stroke. Also, a quarter of those not initially depressed became so six months to one year later.

To elucidate the factors associated with depression, simple correlation coefficients were calculated between a patient's Wakefield score and his score on other scales (Table II). A few patients had to be excluded, as data for them were not available, but most analyses contained over 300 patients. All coefficients are statistically significant ($P<0.01$), with the highest associative factors of items not measuring mood being self-image of recovery ($r=-0.430$) and FAI score at one year ($r=-0.371$). The negative sign indicates that as these scores increase, so less is scored on the Wakefield Depression Inventory (i.e. showing a less depressed mood). Similarly, as functional and intellectual performance improve, showing higher scores, so Wakefield score decreases. However, these coefficients are not high enough to imply necessarily clinical significance; e.g. the Raven's progressive matrices score at six months only accounts for 2% of the variance seen, despite its statistically significant coefficient of $r=-0.146$. Furthermore, the existance of a significant correlation cannot be assumed to indicate a causal relationship.

The strongest correlations are those between measures of mood, either using different instruments (e.g. the Hopelessness scale) or at different times. Wakefield scores are also strongly correlated with patients' self-assessment of mood change from before their stroke.

At three weeks after the stroke there was a significant association ($\chi^2=7.56$, $P<0.05$) between depression and side of stroke symptoms: 75 (42%) of the 178 patients with left-sided symptoms were probably or certainly depressed, compared with 48 (28%) of the 170 patients with right-sided symptoms. Their mean scores were significantly different (left = 13.1, s.d. = 8.6; right = 11.2, s.d. = 7.8; $t=2.17$; $P<0.01$). The mean score (8.3, s.d. = 7.1) of the 31 patients with no unilateral symptoms or signs (e.g. brief or mild aphasia) was significantly lower than in the patients with left-sided symptoms ($t=2.94$, $P<0.01$). There was no significant association between level of depression and laterality of stroke at six months or one year. Patients with aphasia or other communication problems sufficient to preclude assessment – 86 (20%) at one year – were excluded. Most of these patients had right-sided weakness, which complicates interpretation of results.

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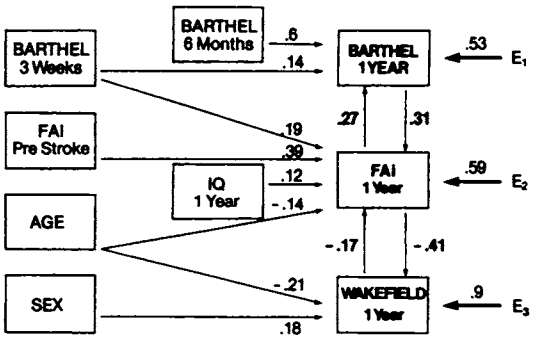


FIG. 1 Path diagram showing influences on depression, social activities and ADL in 348 patients seen one year after stroke.

Women appeared to be significantly more depressed at all three assessment points ($P<0.01$). The number of men/women scoring 19 or more (certain depression) was 38/46 (19%/26%) at three weeks; 29/45 (14%/27%) at six months, and 28/35 (15%/21%) at one year. This sex difference was not simply because more women lived alone: at one year, the mean score of the 45 women living alone was 10.3 (s.d. = 7.2), identical to that of the 27 men living alone. In fact, the difference in depression was most marked in those living with someone: the 180 men had a mean score of 9.7 (s.d. = 7.1), as against 12.0 (s.d. = 7.6) for the 121 women ($t=2.44$, $P<0.02$). The remaining patients were in institutional care. Overall, there was no significant difference in average depression scores between the patients living alone and those living with someone.

Few patients were on treatment for depression six months after the stroke. Information on drugs being taken was available for 274 of the patients assessed, but this information had not been collected for those seen in the early part of the study. Only 23 (8%) were on antidepressant medication; their average Wakefield score was 14.9 (s.d. = 8.0), which was significantly higher ($t=2.35$; $P<0.01$) than the remaining patients (mean = 10.8; s.d. = 8.0). Eleven (48%) had scores of 15 or more, compared with 76 (30%) of the remaining patients, 50 of whom had scores of 19 or more. It seems that most 'depressed' patients were not being treated for depression.

Path analysis (Blalock, 1971) was used to develop a model linking the various factors thought to influence depression: as results were very similar after both six months and one year, only the latter are illustrated in Fig. 1. This technique allows a visual appreciation of the associations between several variables simultaneously. Repeated multiple regression analyses identify the strength of interactions between pairs of variables, while controlling for the effects of the other variables. Path coefficients (i.e. standardised regression coefficients) represent the relative strength of each association.

Figure 1 illustrates the influences on a patient's mood (Wakefield), social function (FAI), and functional ability (Barthel ADL) at one year after the stroke. The arrows to these variables, together with corresponding path coefficients, indicate the relative (but not absolute) strength of influence of static or preceding factors. For example, 'sex' indicates that, when controlling for all other factors, sex has a relatively small influence on depression.

The path diagram further illustrates how a significant correlation, for example that between ADL score and Wakefield score ($r=0.247$), can be 'lost' when controlling for other variables. That is, the path coefficient between ADL and Wakefield scores did not reach a significant level (zero path coefficient), and was therefore ommitted from the diagram. The significant correlation can now be explained by means of an intervening variable – FAI – which may act as a mediator between ADL and depression.

Where reciprocal statistical influences exist between outcome variables, these have been indicated; it is difficult, however, to envisage how social functioning might influence ADL function, and an unidentified intervening variable must be assumed. The consequence of depression in terms of fewer activities (lower FAI score) and vice versa is, on the other hand, more readily understood.

Finally, in understanding the three outcome factors, much of the variance has not been accounted for in terms of the variables measured, so that unmeasured extraneous variables must account for the remainder; 28% of the variance in Barthel ADL is unaccounted for, and extraneous variables are represented by E_1 in the diagram with a path coefficient of 0.53. Similarly, 25% of the variance in FAI (E_2) and 40% of the variance in Wakefield (E_3) are unaccounted for.

Discussion

This analysis confirms that a depressed mood is common after stroke and shows that, although other stroke-induced losses do relate to depression, most depression cannot be explained simply by reference to physical or social disability. One possible explanation is that pre-existing personal or social characteristics may have an important influence on the presence of depression after stroke. It also seems that few depressed patients receive specific medical treatment, despite evidence that tricyclic antidepressants may be effective (Lipsey & Robinson, 1984).

This study included all patients admitted to hospital, but might have missed some of those treated at home; however, our overall incidence of 1.90/1000 per year is not very different from other surveys (aho *et al*, 1980), suggesting relatively complete notification. At one year after stroke, 88% of surviving patients were seen, but 20% of these could not be assessed due to communication problems. Although 'objective' biochemical methods of diagnosing depression have been proposed (Carroll *et al*, 1981), their sensitivity and specificity are still debated (Holden, 1983), while their use in a community study would raise many problems. However, as a wide spectrum of patients was included, with no obvious

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selection according to stroke severity or hospital admission, the sample should be representative of patients suffering stroke but with no communication problems.

The validity of the Wakefield depression scale needs discussion. In this study, the Wakefield score was strongly related to two other measures of depression–the Hopelessness score and the patient's own assessment of his mood change–and it was also related to the prescription of antidepressant medication. We conclude that the Wakefield did measure depressed mood in our patients, although this is not to say that patients with a depressed mood necessarily suffered a depressive illness.

Patients were categorised as 'probably' or 'certainly' depressed because of uncertainty as to cut-off values with an elderly population. Furthermore, it is possible that many 'normal' elderly people are 'depressed': the average Hopelessness score of 5.64 in our study is not significantly different from that recorded in 148 normal people aged over 50 in Dublin (Greene, 1981). Depression may have been present in many patients before their stroke. However, one survey (Bergmann, 1982) of elderly people suggested that 'depressive neurosis' was present in only 3.8% of the 360 tested and 'depressive illness' in 0.6% (all women). These figures are much lower than those seen in our patients with stroke.

Our results confirm the frequency of depression found in other large-scale studies (Robinson & Price, 1982; Robinson *et al*, 1983, 1984*b*; Feibel & Springer, 1982): approximately one-third of patients were depressed at any time after the stroke. We found that the severity of the stroke, as measured by the Barthel ADL scale, did have a direct correlation with depression. One other study (Feibel *et al*, 1979,) noted such a correlation, although the strength of their correlation increased with time, in contrast to the fall we found; other studies (Robinson & Price, 1982; Feibel & Springer, 1982) failed to find any correlation with ADL loss. Two points need to be made concerning this correlation. Firstly, when present, it is relatively small, accounting for 5–15% of all variance; this is probably not clinically important. Secondly, path analysis suggests that non-ADL activities are intervening factors in the relationship between ADL function and depression. We thus confirm the relationship between depression and low social functioning, as previously found (Feibel & Springer, 1982; Feibel *et al*, 1979).

It has not previously been reported that women are more depressed; this difference applied only to those living with someone. Social isolation did not appear to be associated with depression. The other major identified influence on depression at one year was depression seen earlier in the recovery process. Over half of those depressed at three weeks or six months were still depressed at one year. This is a little less than found in another follow-up study, where 10 out of 13 initially depressed patients were still depressed at six months (Robinson *et al*, 1984*b*).

Previous reports on small numbers of selected patients (Robinson *et al*, 1984*a*) have suggested that there is a higher rate of depression in those with lesions in the left frontal region, but the same workers failed to confirm this finding when studying larger groups of patients (Robinson *et al*, 1983). We did not have any information concerning the anatomical location of the stroke, but we have failed to confirm any association between right-sided weakness and depression. Indeed, in the first few weeks those with left-sided weakness were more depressed, although this may be due to exclusion of patients with aphasia. We conclude that even if lesions near the left frontal pole are associated with an increased risk of depression, this cause of depression is overshadowed by other factors in the majority of cases.

This study confirms previous observations that most depression is untreated (Robinson & Price, 1982; Feibel *et al*, 1979,) and also shows that most depression cannot be explained simply as a natural response to physical disability. Indeed, much of the variance in depression is unaccounted for by any of the variables studied, suggesting that other personal or social factors may be important. This study suggests that attempts to reduce depression through improving a patient's social or ADL functioning may not be very successful, although this is not to deny the important association between depression and both low social and ADL functioning.

In conclusion, this study found that a depressed mood was present in about one-quarter of surviving and assessable patients at any time up to one year after stroke. Less than half of those depressed in the early stages were free of depression at one year. The presence of depression was associated with the degree of physical disability, loss of social function, and being female living with someone, but these and other factors measured do not account for much of the depression seen. The path diagram derived from these data gives a broad outline, in visual form, of some important inter-relationships, but will need modification in the light of future research.

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