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DEPRESSED MOOD AND INTRA-HEMISPHERIC LOCATION OF LESION IN RIGHT HEMISPHERE STROKE PATIENTS

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ABSTRACT. **42 stroke patients with verified right hemisphere lesions were studied. Depressed mood was measured by means of a global rating scale, and an inventory administered as a structured interview. As measured by the global ratings and one of the inventory subscales, patients with deep, retrorolandic lesions showed significantly more depressed mood than other patients. The findings are discussed in the light of current relevant research, and implications for management of depressed mood in stroke patients are suggested.**

Key words: stroke, right hemisphere, depression.

Emotional behaviour after unilateral cerebral lesions has often been described in terms of catastrophic reactions following left hemisphere (LH) lesions and emotional indifference after right hemisphere (RH) affection (11). In several studies, depression is found to occur more frequently after LH than RH damage (3, 18, 19) but a depressed mood has also been found after RH stroke (10) and other RH affections (15). Tucker (21), Kinsbourne & Bemporad (13), Finset (6) and others have reviewed this literature.

There are indicators that depressed mood may interfere with the rehabilitation process. Unpublished findings from our own studies indicate that depressed mood has significant effect on rehabilitation outcome upon discharge from the rehabilitation hospital. (8), and other more informal clinical data confirm this conception.

Recently, the degree and type of depressed mood after stroke have been related to *caudality* of lesion. Robinson and co-workers have in several studies (18, 19) found depressed mood to be most severe and frequent after left anterior damage. Among patients with RH damage, those with a posterior lesion tended to be more depressed than right frontal patients. Finset (4) found in a preliminary study of RH stroke patients a relationship between depressed mood and caudality in the same direction. In a recent study Sinyor et al. (20) also found an association between depression and lesion location close to the frontal pole in LH patients, but not clear enough to reach statistical significance. In his RH sample both patients with clear-cut posterior lesions and patients with lesions close to the frontal pole tended to have high depression scores.

In the present study, which is part of a research program investigating a broad range of neuropsychological deficits after RH stroke (7), depressed mood in RH stroke patients is studied with reference both to caudality and depth of the lesion.

METHODS

Sample. The subjects of the present study are 42 stroke patients admitted to Sunnaas Rehabilitation Hospital for inpatient rehabilitation. All patients had a diagnosed CVA with RH lesion confirmed by CT-scan, and with onset within 6 months before neuropsychological testing. Patients with left handedness, signs of LH affection, and clinical signs of aphasia were excluded. Patients consecutively admitted to the hospital, who satisfied these criteria, and went through the examination program of the study, were included in the sample (except one patient with perirolandic lesion difficult to classify according to our lesion categorization system (9).

The sample includes 31 male and 11 female patients, with a mean age of 60.1 years at admission, in a range from 43 to 78 years. Patients were neuropsychologically tested within 26 weeks after the CVA (17 within 6 weeks, 15 between 6 and 12 weeks, and the remaining 9 patients between 12 and 26 weeks post CVA). 37 of the patients suffered from a cerebral infarction due to thrombosis or embolism. The remaining 5 patients suffered from cerebral or subarachnoid haemorrhages.

Location of lesion. CT scans were analyzed by a radiologist, (M. H.), who had no knowledge of the depression data or any other test- or rating-scores in the study. The caudality and depth of the lesion were classified into one of four categories:

(1) patients with lesions restricted to right subcortical regions, such as the internal capsule (*capsular* lesions), (2) patients with lesions restricted to or mainly affecting the prerolandic, *anterior,* part of RH, and (3) patients with lesions restricted to or mainly affecting the postrolandic, *posterior* part of RH. The latter category was subdivided

Table I. *Criteria for depressed mood applied in the global rating scale*

1. No indication of depressed mood
2. Some feeling of sadness and low spirits, within normal range
3. Depressed mood and definite feelings of sadness, experiences as a serious problem for individual or family, may be within the concept of a normal grief reaction
4. Definite pathological depression
5. Pervasive feelings of sadness, meaninglessness, the depression dominates totally

Table III. *Number of patients depressed and not depressed in anterior and posterior lesion categories (percentages in parentheses)*

	De-pressed mood	Not de-pressed mood	Total
Capsular lesion only	3 (21)	11 (79)	14 (100)
Mainly anterior lesion	2 (25)	6 (75)	8 (100)
Mainly posterior lesion	10 (50)	10 (50)	20 (100)
Total	15 (36)	27 (64)	42 (100)

into patients with *superficial cortical posterior* lesions, mainly over the parietal convexity of the hemisphere; and *central* lesions, most of them extending deep in the hemisphere.

Measures of depressed mood. The following measures of depressed mood were applied in the study:

(1) *A global, clinical rating of depressed mood.* All patients in the sample are rated acccording to a five point rating scale, designed especially for the present study. The five values of the scale are specified in Table II. Patients were rated by a psychologist (A. F., L. G., or N. I. L.) as a part of the standard neuropsychological examination during the first weeks of the patient's stay. Routines were arranged so that the psychologist had no knowledge of data on lesion location at the time of testing (Table I).

(2) *A revised version of the ADI inventory.* The ADI inventory (Anxiety–Depression–Irritability), designed by Havik & Mæland (12), is a 32 item, 5 subscale inventory aimed at assessing emotional reactions to somatic illness. The inventory was revised for the present study. Items were reformulated to allow administration as a structured interview (as some patients, most frequently due to spatial neglect, had reading difficulties), and a few items were taken away. Two new five item subscales (not reported in this paper) were constructed (7). The inventory has two subscales for depressive affect: the Somatic symptoms of depression and Psychological symptoms of depression, each consisting of five items.

Some of the patients were rated with selected items from *CPRS (Comprehensive Psychiatric Rating Scale).* The CPRS is a 67 item rating scale, developed by British and Swedish psychiatrists, psychologists and clinical pharmacologists, specially designed to cover psychopathological variables likely to be changed by treatment (1). Ten items were chosen for the rating of depressive symptomatology (14). CPRS was entered as a rating procedure during the course of the study. Only 29 patients are CPRS rated. Patients were rated by a trained psychologist.

Table II. *Frequency of depression based on global rating scale*

	N	%
1. No depression	11	(26)
2. Some sadness, mildly depressed mood	16	(38)
3. Definite depressed mood, problem for individual	9	(21)
4. Pathological depression	4	(10)
5. Pervasive and totally dominating depressed mood	2	(5)
Total	42	(100)
Categories 3–5	15	(36)
Categories 4–5	6	(14)

RESULTS

Frequency of depressed mood. The assessment of incidence of depressed mood after RH stroke will obviously depend on what level of depressive affect that is taken as cut off criterion. 15 patients (36%) show a consistent depressed mood, express depressive affect, and seem to suffer from this depressed mood to an extent that represents a probable hindrance to their rehabilitation, rating "3" on the global rating scale. In a larger sample of patients with left hemiplegia including many patients without CT scans in addition to those included in the present study, Finset & Haakonsen (8) found 44% of the patients to be depressed according to the same criterion. Only 6 patients (14%) are depressed to the extent that they show a pathological depression in a stricter psychiatric sense (Table II). In the larger sample, 21% of the patients were depressed to that degree.

Depressed mood and location of lesion. Based on findings by Robinson et al. (18, 19) and our own preliminary analysis (4), we related degree of depressive affect to caudality of lesion according to a simple mainly posterior vs. mainly anterior categorization. Patients with capsular strokes were in-

Table IV. *Number of patients depressed and not depressed in central posterior and all other lesion location categories compared (percentages in parentheses)*

	Depressed mood	Not depressed	Total
Central posterior lesion	8	5	13
Other locations	7	22	29
Total	15	27	42

$X^2=5.48$. df=1. $p<0.02$.

cluded in a separate category. As shown in Table III, the percentage of patients rated to show a depressed mood somewhat larger in the posterior group than in other categories, but not to a degree that is statistically significant.

When data were more closely analyzed, it became evident that the seven patients with superficial posterior lesions most often over the parietal convexity of the hemisphere deviated from the other patients with posterior lesions in as much as most of them tended to display very little, if any, depressive affect. Only two of seven patients in the superficial posterior category were depressed, as compared to 8 of the 13 remaining posterior patients. Moreover, three of the seven patients with superficial posterior lesions showed no indication of depressed mood at all, whereas none of the patients with central and deep posterior lesion were rated in the "No indications of depressed mood"-category. Thus scores on the depressed mood measures were broken down in four lesions location categories, differentiating between patients with capsular lesions, patients with lesions mainly of anterior regions of the hemisphere, patients with lesions encompassing central and most often deep areas posterior, and finally patients with superficial posterior lesions, an interesting pattern appeared in the findings.

(1). *Global ratings*. In the central posterior category, 8 patients were rated to show depression according to the cut of point of "3" at the rating scale (62%). This was the only lesion category where the majority of the patients showed significant depressed mood (values "3" to "5" on the rating scale).

In all the other groups, the number of patients rated to be depressed tended to be around one fourth of the patients in the category; 2 patients with superficial posterior lesions (28%), 2 patients with mainly anterior lesions (25%) and 3 patients with capsular lesions (21%) showed significant depressed mood.

As the numbers are so small, and the trend so consistent, we have taken the freedom to cross-tabulate patients in the only lesion category with a majority of depressed patients—the central posterior category—against all other patients (Table IV).

As mentioned above, 11 of the 13 central posterior patients had lesions extending deep in the hemisphere. Seven of these 11 patients displayed significant depressed mood (64%).

(2) *ADI inventory scores*. Patients in the *central, mainly posterior* lesion category show significantly more depressed mood, measured by the psychological symptoms subscale (Table V). The means of patients with central anterior lesions are around the total sample mean value, whereas the mean scores of superficial posterior and capsular patients are below the sample mean. Also in the reporting of somatic symptoms of depression the patients with a central, mainly posterior lesion are the most depressed, but only by the psychological symptoms subscale the findings reach a level of statistical significance.

Unfortunately, only 29 patients are rated with the CPRS. The trends are as in the global ratings, that patients with central, mainly posterior lesions are most depressed, whereas patients with pure posterior lesions are least depressed. Because of the small number of patients, the trend does not reach statistical significance.

DISCUSSION

The findings of the present study confirm the view that depressed mood may occur also among patients with RH stroke, and that the occurrence of this symptom seems to be related to intra-hemispheric location of lesion. The findings do, however, indicate a more complicated mechanism involved than presumed from earlier indications of a posterior–anterior dimension (4, 19).

The findings also differ from a recent study by Sinyor et al., (20) attempting to replicate the work of Robinson et al. Sinyor et al. find a curvilinear relationship to depression in RH patients, with both anterior and posterior patients tending to be depressed. Both samples are small (16 RH patients in the study by Sinyor et al., and CT data are analyzed

Table V. *ADI Inventory scores of depressed mood broken down against lesion locations*

Location category	ADI-psychol. M	ADI-psychol. S	ADI-somatic M	ADI-somatic S	ADI-total M	ADI-total S	N
Capsular lesion only	9.6	3.8	10.3	4.4	19.9	5.6	14
Mainly anterior	12.4	4.8	11.5	4.8	24.3	6.9	7
Central, mainly posterior	15.7	6.4	12.2	5.5	27.8	10.9	13
Cortical, pure posterior	10.6	5.9	9.0	3.6	19.6	8.8	7
Total	12.3	5.8	10.9	4.8	23.1	8.5	41
	F=3.40 P<.05		F=1.00 NS		F=2.75 NS		

differently in the two studies. Depth of lesions, proving to be a crucial variable in our study, is e.g. not addressed in the paper of Sinyor et al.

Taking into account the by now consistent finding that depressive affect is associated with frontal pathology if the lesion is in the left hemisphere, our findings are not easy to interpret. Robinson et al. have found asymmetries in NA (noradrenalin) balance in rats, that partly may explain laterality differences, but hardly the caudality effects. Asymmetries in neurotransmitter function recently reported as suggested by Tucker & Williamson (12) as well as asymmetries in cortico-limbic connections (Bear, 1986) may also be relevant factors in explaining differences in emotional behaviour after lateralized lesions.

Kinsbourne & Bemporad (13) and Finset (5) suggested that the *diagonal* pattern in depressive affect after unilateral lesions (i.e. the trend to find depressed mood after *left anterior* and *right posterior* lesions, areas that are located along a diagonal axis in the brain) indicates the existence of intrahemispheric and/or interhemispheric release and inhibitory mechanisms. Evidence from several lines of research indicates a special role of the right frontal lobe in depression (21). One might speculate that a lesion in the neigbouring area (anterior LH or posterior RH) somehow affects the right frontal lobe in a way relevant for depressed mood. Or the other way around: a frontal RH lesion might suppress a depressed mood otherwise exhibited.

It has been suggested that the depressed mood seen in RH stroke patients may differ qualitatively from depressed mood after LH stroke.

Whereas the depressed LH patient often exhibits anxiety, the depressed RH patient most typically seems to suffer simply from a lowered mood level, with few other clinical signs of depression, also compared with depressed patients without brain damage. These differences, observed in the nature of depressed mood after RH and LH lesions (5, 13), may indicate that what we call depressed mood in both cases may be different phenomena.

Any proposed explanation of changes in emotional behavior after posterior RH damage should also take into consideration the proposed important role of that part of the brain in processing emotionally loaded stimuli. A dysfunction in emotional behaviour after a right posterior lesion could at least in part be due to the failure of the brain to decode the meaning of an emotional message (6).

Both the relationships to intra-hemispheric location and the differences from other depressive reactions, indicate the organic nature of the depressed mood associated with RH damage. This conception of RH depressed mood has important clinical implications for the rehabilitation of RH stroke patients.

If the depressed mood after RH stroke were to be considered as solely a natural reaction brought about as a reaction to disability, a conventional psychotherapeutic approach aiming at coming to terms with the underlying crisis should be tried. Moreover, antidepressants would probably be of little use. The rehabilitation literature is sparse in evaluating such an approach.

The view presented in this study, that depressed mood in RH stroke patients has an organic component, implicates that the patients are vulnerable to more or less wrongfully view their depressed mood as a psychological rather than organic consequence of the strokes (or other cerebral insult) they have recently suffered. We have suggested that an adequate psychotherapeutic response to RH depressed mood would be a *reattribution therapy* approach (5, 6).

The term "reattribution therapy" is taken from cognitive-behavioral therapy (17). It implies an effort on the part of the therapist to help the patient correct wrongful conceptions (misattributions) concerning him- or herself. In our case, an early explanation to the patients that depression is a very common concomitant symptom in persons with that particular lesion may prevent them from being too absorbed by depressed thoughts. The cognitive content of therapy would be a reattribution rather

than emotional working through and emphasize (and preferably make provisions for) physical and mental activity.

Antidepressant drugs may be indicated. Lipsey et al. (16) administered a double blind study to try out nortriptyline to stroke patients. The treated group showed significantly better remission than the placebo group, both in LH and RH patients. We (5) have in an unpublished pilot-study with 14 depressed RH patients, using doxepin (Nozinan), observed a corresponding statistically significant better remission rate in the treated group.

The research in the area of emotional behaviour after lateralized lesions is as of no way too sparse to allow comprehensive explanations of the findings reported in the present study. Although findings from Robinson et al.'s (18, 19), Sinyor et al.'s (20) and our studies differ in their conclusions, even Sinyor et al. (who found no significant intrahemispheric differences in the RH sample) concedes that depressed mood even after RH damage seems related to locus of lesion. Future research may sort out the effects of such different mechanisms as neurotransmitter asymmetry, separate cortico-limbic pathways, intra- and interhemispheric interaction effects, and specific effects of the localization of different elements of emotional processing.

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