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COGNITIVE DISORDER AND DEPRESSION
(An Analysis of the Causal Relationship and Susceptibility to Relapses)*

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SUMMARY

A prospective study of 45 cases of unipolar depression was conducted with the aim to examine the hypothesis that cognitive disorder predisposes to depression. The data indicated that the absence of the cognitive disorder during remission was statistically significant in 41 cases who remitted. Of the latter, 27 were free from cognitive disorder, while 14 continued with it. This fails to support the causal role of the cognitive disorder. It was also observed that those with persisting cognitive disorder in remission ran the risk of early relapse. It is suggested that persistence of cognitive disorder in remission could be a predictor of early relapse and offers the possibility of using cognitive behavioural therapy for such select cases.

Nearly 25 years ago, Beck (1963, 1964) through clinical observation, demonstrated that thinking disorder was basic to Depression and it was in the nature of "intrusive thoughts" and "cognitive distortions". From these, Beck proceeded to identify the cognitive errors (selective abstraction, arbitrary *inference, over-generalization, maximization* or minimization) and the cognitive triad (negative perception of self, the world around and the future) as the predisposers to disease. Underlying these errors are the cognitive schemas. The cognitive errors and triad were endowed with a causal role and accordingly depression came to be interpreted as the result of lifelong habit of self deprecation. Cognitive theory grew out of discontent with the psycho-analytical school's preoccupation with the contents and mechanisms of the 'unconscious' and also with behaviour therapy which ignored the individual's subjective thought content. Abnormal biochemistry too was found to be inadequate since it excluded the lesion from the individual's awareness. On the other hand, with the cognitive hypothesis "man has the key to understanding and solving his psychological disturbances within the scope of his awareness" (Beck, 1976). A critical assessment of the cognitive theory of causation of depression is now due. Cognitive theory it appears argues against "post hoc ergo propter hoc" argument namely because B (thought disorder) follows A (depressed mood), then A must have caused B. Bemporad (1980) suspects that the disordered thinking in depression may be consequent to depression and not *vice versa*. Hamilton and Abramson (1983) on comparing depressed and non depressed psychiatric patients found the thought abnormalities in the former but not in the latter. The thinking disturbances described by Beck were observed in depressed patients when compared with those with low back pain (Lefebvre, 1981). The occurrence of 'intrusive thoughts' in depression has

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been confirmed by Hollon and Kendall (1980).

Hopelessness and helplessness also characterize depressive thinking and are closely related to Beck's cognitive triad. Hopelessness and helplessness have been recorded also in neurotic (Frank, 1961) as well as in psychosomatic disorders (Engel, 1962). Cognitive disorders perse may not cause depressive illness. Any stressful event or a biochemical disorder may activate the underlying cognitive schemas precipitating clinical depression. For example a biochemical abnormality may initiate depression with the symptom of thinking disorder giving fresh life to the mood in the manner of vicious circle. The theory requires that cognitive disorder should be present prior to the occurrence of depressive episode and 'continue' following recovery or remission. This latter explains the recurrences. The absence of cognitive disturbance during remission or recovery renders the cognitive theory untenable with regard to causation. To explain this, it was suggested that the cognitive disorder may be 'latent' under these circumstances. It is paradoxical that the 'unconscious', is resorted to while the cognitive theory itself arose as a move away from this psycho-analytical concept. It looks as though it will take a longer time to establish the validity or otherwise of cognitive causation of depression. "It is not yet easy to assess the place of Beck's work : it has not reached a final point of equilibrium" (Paykel, 1987).

AIM AND OBJECTIVES

The principal objective of the present study was to record the cognitive disorder in the active phase of depressive disease and to examine for its persistence during remission. To study the relationship between the persistence or otherwise of cognitive disorder in these remissions and the subsequent recurrence of depression was the other aim. The overall aim was to test the theoretical soundness of Beck's hypothesis that cognitive disorder is causal to depression.

MATERIAL AND METHODS

Fifty consecutive hospitalised cases (M = 28; F = 22) of unipolar depression—major (DSM III) from the Institute of Psychiatry, Government Rajaji Hospital, Madurai Medical College, Madurai, during the period November 1986 and April 1987 formed the material for the study. The ages of the subjects ranged from 15-76 years. The duration of their index episodes varied from less than 3 months to more than a year. Twenty seven were married, 13 were single and the rest widowed, divorced or separated. In thirty patients, episode of depression was the first one, while among others, eleven had one, two each had two and two had three and five had four or more episodes prior to the index examination. A history of manic episode prior to intake or during the period of follow-up and/or symptomatic depression were the exclusion criteria. The diagnosis was arrived at by two psychiatrists of the team (AVR, TKR and ERP) independently. Excluded from the study were five cases, for reasons of turning bipolar (N = 1) or revision of diagnosis (N = 3) or drop out (N = 1). The severity of depression was rated by Beck's Depression Inventory (BDI). The data on cognitive disorder were recorded by Beck's Hopelessness Scale (BHS), Crandell's Cognitions Inventory (CCI) and on eight items of Beck's Depression Inventory (Pessimism, Sense of failure, Guilt feelings, Self-hate, Self accusations, Self punitive wishes, Social withdrawal and Body image).

OBSERVATIONS

Index Status and First Remission

At the time of intake 25 subjects were rated on BDI as severe, 18 'Moderate Severe' and one each as 'Mild-Moderate' and 'Mild'. On BHS, 28 cases were rated as 'Severe',

9 'Moderate' and 8 'Mild'. On Crandell's Cognitions Inventory, all the subjects showed high rating on all the four dimensions of the test (detachment, hopelessness, helplessness and self rated inferiority). On the eight items of the BDI, all the subjects scored for their presence. Standard therapy with antidepressants and wherever indicated combined with ECT was administered to all the subjects. They were reporting at regular intervals at the 'Depression Clinic' of ICMR centre for Advanced Research on 'Health and Behaviour', Government Rajaji Hospital, Madurai. The research protocol required every 4th monthly follow-up. Fortyone cases showed remission following treatment, while four continued in an unchanged state. The quality of remission was assessed by clinical examination and by BDI rating (criterion of 0-9). The tests for cognitive disorder were repeated in these remitted cases by administration of BHS, CCI and noting the 8 items of BDI. Among the remitted subjects, 27 were free from cognitive disorder while in 14 they persisted. All the remitted cases, scored within normal on BDI and hence, those who scored 'one' on any of the 8 items have been reckoned as indicative of cognitive disorder. The scoring of 'zero' on any of 8 items was recorded as absence of cognitive disorder. The data in respect of the BDI (severity), BHS, CCI and 8 items of BDI (cognitive disorder) at the point of intake and in first remission are offered in Tables I and II.

First Relapse (N = 8) and Second Remission (N = 6)

Eight cases relapsed following remission during the follow-up period of one year from intake. A standard clinical examination was repeated on them along with the administration of the BDI (severity), BHS, CCI and noting the eight items of BDI. All the relapsed subjects rated high on BDI as well as demonstrating the presence of cognitive disorder. With continuation of treatment, six of them showed remission for second time. In one, the illness continued unchanged. There was one case of mortality (Suicide ?). Repetition of clinical examination and administration of all the schedules in these six remitted cases (BDI 0-9) revealed a persistence of cognitive disorder in five subjects and its absence in one. Their rating on BDI was nil or minimal (0-9). Six among these eight relapsers were drawn from the sub group of 14, who had cognitive disorder and 2 from the

TABLE I. *Data on Index Evaluation BDI Rating (N = 41)*

BDI Rating	Number	%
Normal	—	—
Mild	—	—
Mild Moderate	1	2.4
Moderate Severe	17	41.5
Severe	23	56.1
Mean Severity	32.1	
S. D.	8.5	
Range	18–53	

Cognitions Rating (N = 41)

Cognitions	BHS Rating	CCI Rating			
		Detachment	Self rated inferiority	Helplessness	Hopelessness
Nil/Minimal	—	2	—	1	1
Mild/ Moderate/ Severe	41	39	41	40	40

8 Cognitive Items on BDI (N = 41)

Cognitive Items	Number	%
Present	41	100.0
Absent	—	—
Mean Severity	11.0	
S. D.	3.9	

TABLE II. *First Remission BDI Rating (N=41)*

BDI Rating	Number	%
Normal	41	100.0
Mild	—	—
Mild Moderate	—	—
Moderate Severe	—	—
Severe	—	—
Mean Severity	4.5	
S. D.	3.3	
Range	0.9	

Cognitions Rating (N=41)

Cognitions Rating	BHS Rating	CCI Rating: Detachment	Self rated inferiority	Helplessness	Hopelessness
Nil/Minimal	27	27	21	30	29
Mild/Moderate/Severe	14	14	20	11	12

8 Items on BDI (N=41)

Cognitive Items	Number	%
Present	19	46.3
Absent	22	53.7
Mean Severity	0.73	
S. D.	1.0	

other group of 27 without such disorder in their first remission. The other six in first remission with persistence of cognitive disturbances were followed up and were found to be experiencing transitory depressive states (not amounting to clinical depression) in response to life events. Their BDI ratings were within normal limits, but with elevated ratings on BHS, CCI and 8 items of BDI. Data on first relapse, second remission and second relapse are shown in the Table III, IV and V.

TABLE III. *Data on First Relapse (N=8)*

BDI Rating	Number	%
Normal	—	—
Mild	—	—
Mild Moderate	1	12.5
Moderate Severe	5	62.5
Severe	2	25.0
Mean Severity	27.1	
S. D.	2.7	
Range	22.30	

Cognitions Rating (N=8)

Cognitions Rating	BHS Rating	CCI Rating: Detachment	Self rated inferiority	Helplessness	Hopelessness
Nil/Minimal	—	—	—	—	—
Mild/Moderate/Severe	8	8	8	8	8

8 Cognitive Items on BDI (N=8)

Cognitive Items	Number	%
Present	8	100.0
Absent	—	—

Unchanged course (N=5)

At the time of the preparation of this report, there were 5 subjects pursuing an unchanged course, four from the time of intake and one following the first relapse. The severity of depression (BDI ratings) has dropped but not to within the range of normal but yet with persisting evidence of cognitive disorder.

DISCUSSION

It is difficult to obtain a 'preknowledge' of the cognitive disorder in an individual antedating the first episode of depression. Al-

TABLE IV. *Data on II Remission (N=6) BDI Rating*

BDI Rating	Number	%
Normal	6	100.0
Mild	—	—
Mild Moderate	—	—
Moderate Severe	—	—
Severe	—	—
Mean Severity	3.8	
S. D.	2.4	
Range	0.7	

Cognitions Rating (N=6)

Cognitions Rating	BHS Rating	CC Rating: Detachment	Self rated inferiority	Helplessness	Hopelessness
Nil/Minimal	5	1	2	2	2
Mild/Moderate/Severe	1	5	4	4	4

8 Items on BDI (N=6)

Cognitive Items	Number	%
Present	4	66.7
Absent	4	33.3

TABLE V. *Data on II Relapse (N=1)*

Factor	Rating
I BDI	Mild Moderate
II BHS	Severe
III CCI	
(a) Detachment	Moderate
(b) Self-rated inferiority	Moderate
(c) Helplessness	Moderate
(d) Hopelessness	Mild

ternatively, one could examine for the presence of such a disorder in the recovered or remitted cases of depression. The present study revealed that out of 41 subjects who remitted following treatment, 27 were free from cognitive disorder while 14 continued to have them. The difference between these two subgroups of remitted depressives in terms of absence of cognitive disorder is statistically significant (Table VI).

TABLE VI. *Cognitions Evaluation During I Remission (N=41)*

Item (During Remission)	Number rated as: Nil/Minimal	Mild/Moderate Severe	X^2	P
BHS—Hopelessness	27	14	4.12	0.05
Crandell Cognitions Inventory				
Detachment	27	14	4.12	0.05
Self-rated inferiority	21	20	0.02	NS
Helplessness	30	11	8.8	0.01
Hopelessness	29	12	7.0	0.01

This observation fails to support the causal role of cognitive disorder. That the cognitive disorder in states of remission from depression did not appreciably differ from the normal subjects has been recently commented upon by Gelder (1985). It is possible that what was described as a 'trait' by cognitive theorist may be a 'state' feature'. However, the persistence of cognitive disorder in remitted cases in our series appears to have a bearing on the susceptibility to early relapse. Of the eight patients who suffered first relapse, six were drawn from the group of remitted cases with persisting cognitive disorder and only two from those without the disorder. It indicates that the cognitive disorder in remission may be a risk factor for early relapse. The difference between the relapsed patients in respect of their cognitive status during the preceding remission is significant (Table VII).

TABLE VII. *Data on I Relapse (N=8)*

Item	No. Rated in Previous Remissions		X^2	P
	Nil/ Minimal	Mild/ Moderate/ Severe		
BHS—Hopelessness	2	6	5.29	0.05
CCI Self-rated inferiority	1	7	4.19	0.05
Helplessness	1	7	15.0	0.001
Hopelessness	2	6	5.29	0.05
Detachment	2	6	5.29	0.05

A study of second remission following the first relapse revealed 5 subjects continuing to retain cognitive disorder with only one being free from it. There were again 2 relapses for the second time from among the former. The findings at this stage of the study show that persistence of cognitive disorder in remission may be a predictor of early relapses. It may be argued that these patients with persistent cognitive disorder had not struck remission and this thought disorder was part of incomplete remission. However, clinical examination and BDI ratings revealed them to be in remission.

The relapses were analysed on the variables like age, sex, number of relapses prior to the index assessment and the degree of severity of illness at index examination. A positive relationship was observable on the sex variable only with females outnumbering males (2M; 6F). There was no relationship between the relapses and the other variables. The follow-up study of 6 first remissions who did not develop recurrence of diagnosable depression revealed elevation of ratings on BHS and CCI with day to day events in life. This again indicates that remitted depressives with persisting cognitive disorder are also susceptable to accentuation of their cognitive disturbances. Contrarily, the follow-up of 27 remitted depressives without cognitive disorder revealed only two relapses who again remitted. Thus a differing course of depression in the two sub-groups namely those with the persistence of cognitive disorder and those without is evident during one year follow-up. It is likely that cognitive behavioural psychotherapy may be directed towards this group of patients with a view to prevention of relapse. That cognitive behavioural therapy would prevent relapse is yet to be demonstrated (Gelder, 1985).

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