MEMORY IN DEPRESSION

S. CHANDRA, M.D. (Psych.), D.M.R.E.
A.K. AGARWAL, M.D. (Psych.), D.P.M.

SUMMARY

Out of 88 cases of depression, admitted in Department of Psychiatry, K.G.'s. Medical College, Lucknow, 33 cases fulfilling the inclusion criteria were assessed for severity of depression and memory performance and were compared with memory scores of normal healthy controls. After treatment with drugs, improved depressives served as their own controls also. Memory scores of depressives were found to be significantly poor than those of controls and of their own after recovery. Depression was found to be negatively correlated with memory. Mental control subfunction of memory was most sensitive for depression. Probable mechanism of memory disturbances were discussed.

Memory disturbances are well known in psychiatric patients since long. In Charak Sambita (1948) it is described that "When on account of the mind being clouded with passion and delusion, retention of free knowledge is destroyed and is called derangement of memory". Dysnesia is accepted as diagnostic feature of acute as well as chronic psycho-organic syndromes. There is, however, no unanimity of opinion regarding memory disturbances in depression. "There is no clouding of consciousness, no disorientation, no defect of memory or intellect" in depression (Batchelor, 1969), while Shader and Salzman (1978) are of the view that depression of mood often precedes the loss of memory. Elderly depressives many a times, manifest memory impairment of such proportion so as to enable them to be included under the label of pseudodementia (Wells, 1979).

Cronholm & Ottosson (1961) compared 20 depressives out of group of 45 with controls, matched for age, education and sex, as regards their memory performance. Immediate and delayed reproduction (learning and retention) were significantly low in depressed patients. 42 cases out of group of 45, who responded after E.C.T. showed improvement in immediate reproduction and slight improvement in delayed performance. Friedman (1964), however, could find significant different values in only 9 out of 82 test scores, when 2 groups of subjects with and without depression, of same age, education, sex, marital status, nationality, religion and intelligence level were compared. Kahn et al. (1975) while investigating memory function in depressed patients and non-patients aged 50 years or more found that subjective complaints of memory impairment correlated with severity of depression but there was little relationship with objective memory performance. Sternberg & Zarvik (1976) compared 26 depressives with matched controls as regards memory function for short term and long term memory, and obtained marked impairment in short term memory without impairment of long term memory. 20 recovered depressives served as their own controls. It was observed that greater the improvement of clinical state the greater was improvement in short term memory. Long term memory was not influenced by the therapeutic success.

Several workers (Cronholm & Molander, 1964; Zamora & Kebelbing, 1965; Post, 1966; Zinkin & Birtchnell, 1968; Holiday et al., 1968) observed post

1. M.O. Incharge, Mental Health Clinic, G.S.V.M. Medical College, Kanpur.
2. Reader, Department of Psychiatry, K.G.'s Medical College, Lucknow.
E.C.T. improvement in memory functions in recovered depressives in spite of the dysmnesic effect of E.C.T. These workers presumed that pre-ECT lowering of MQ scores were because of depressed state. Levy (1968) in a carefully conducted study found reduction in post-ECT scores on WMS by an average of 14 even after significant reduction in severity of depression. Kendrick and Post (1967) compared memory performance of four groups of patients. Depressives scoring on synonym learning test in brain damage range were dropped. No difference was observed between depressed patients and normal subjects in the scores on WAIS verbal and performance, synonym learning, paired associate learning and digit copying tests. Strongren (1977) investigated influence of depression on memory in 152 patients suffering from depression (endogenous) and receiving E.C.T. It was concluded that depression impairs memory function as measured by WMS and tests of retention and recovery from depression eliminates the impairment of memory. Mental control sub-function was slightly more sensitive than other memory sub-functions, while depressive appearance and agitation factors of depression were found to be significantly effecting the memory.

Thus, in view of the contradictory and inconclusive reports regarding effect of depression on memory, it was proposed to undertake the present study with the aims of assessing,

(a) Effect of degree of depression on memory function,

(b) Relationship between change in depression and change in memory with treatment,

(c) The effect of depression and its various factors upon memory and its various sub-functions.

MATERIAL AND METHODS

The sample of study was selected from subjects admitted with diagnosis of depression in Department of Psychiatry, King George's Medical College, Lucknow. Only those cases, who fulfilled the criteria for Primary depression as laid down by Winokur (1973), were included in study. Other inclusion criteria adopted for selection of sample were: age between 20-45 years; minimum education up to 8th class; no E.C.T. within last 6 months; and subjects scoring not more than 22 on Taylor's Manifest Anxiety Scale (TMAS) (Taylor, 1953).

All subjects were kept drug free for a period of one week before assessment. Subjects were rated on TMAS and Beck's Depression Inventory (BDI) (Beck et al., 1961). Their memory performance was assessed on Weschler memory scale (WMS) form II (Weschler, 1945). Test administration was done during two sessions with a break of nearly half an hour. The evaluation of all patients was uniformly done in the afternoon in order to negate the effect of diurnal variation in symptoms.

After putting patients on tricyclic antidepressants, a weekly clinical evaluation was done. As soon as patients were found to be free of depressive symptomatology on mental status examination, BDI was again administered and if scores on BDI were found 15 or below, patients were subjected to assessment on WMS. If, however, BDI scores turned out to be 16 or higher, memory assessment was postponed for one or more weeks, until patients recovered from depression, as evident from BDI score of 15 or below. There was an interval of minimum 24 days between 1st and 2nd memory assessment.

33 Normal subjects drawn from available relatives of patients, group-matched for age, sex and education having no history of physical or mental illness and scoring 9 or less on BDI and <23 on TMAS constituted control group. They were administered WMS for assessment of their memory.

To identify which subfunction of memory is most influenced by depression
and its various factors, three subfunction of memory and three factors of depression were tested. The subfunctions of memory were:

- Mental Control (Current information + Orientation + Mental Control + Digit Forward),
- Verbal learning (Logical memory + Associate learning + Digit forward),
- and visual reproduction.

Mental control represents readily accessible matter while verbal learning requires some elaboration of material. Visual reproduction measures non-verbal memory.

Three factors of depression as obtained by factor analysis of Weckowicz et al. (1967) were guilty depression, retarded depression and somatic disturbance. Guilty depression includes following items of BDI—Guilt feeling, sense of punishment, self accusation sense of failure, self punitive-ness, self hate, depressive mood, indecisiveness and pessimism. In this combination of symptoms the experience of sadness and guilt is predominant and the higher mental processes relating to self-concept and the meaning of personal existence seem to be involved. Retarded depression is combination of following items : work inhibition, fatigu, lack of satisfaction, depressed mood, somatic pre-occupation and indecisiveness. Three items of BDI constituted somatic disturbance—weightloss, loss of appetite and sleep disturbance.

RESULTS

Socio-demographic variables of sample show that majority of experimental subjects (48.5%) belonged to 40-45 years age group while youngest age group (20-24 years) was represented by only 6.1% of cases. Educationally the sample largely belonged to lower educated group (less than high school 48.5%). Mean age of experimental and control group was 36.9 years and 35.3 years respectively, difference being not statistically significant. On other variables also experimental and control groups were quite similar to each other.

| TABLE-I. Socio-demographic Variable of Sample | Experimental Group (N=33) | Control Group (N=32) |
| Age (in years) | N % | N % |
| 20—24 | 2 | 6.1 | 2 | 6.1 |
| 25—29 | 4 | 12.1 | 4 | 12.1 |
| 30—34 | 7 | 21.2 | 7 | 21.2 |
| 35—39 | 4 | 12.1 | 4 | 12.1 |
| 40—45 | 16 | 48.5 | 16 | 48.5 |
| Range | 20-44 years | 21-44 years |
| Mean | 36.9±6.5 | 35.3±8.9 |
| Education | | |
| Less than High School | 16 | 48.5 | 16 | 48.5 |
| High School to Intermediate | 10 | 30.3 | 10 | 30.3 |
| Graduate and Post graduate | 5 | 15.1 | 5 | 15.1 |
| Technical Education | 2 | 6.1 | 2 | 6.1 |
| Family Structure | | |
| Unitary | 14 | 42.4 | 12 | 36.4 |
| Joint | 19 | 57.6 | 21 | 63.6 |
| Religion | | |
| Hindu | 28 | 84.9 | 27 | 81.8 |
| Muslim | 5 | 15.1 | 6 | 18.2 |
| Occupational Status | | |
| Labour | 6 | 18.2 | 8 | 24.2 |
| Business | 7 | 21.2 | 5 | 15.3 |
| Service | 16 | 48.5 | 17 | 51.5 |
| House wife | 4 | 12.1 | 3 | 9.1 |
| Domicile | | |
| Rural | 14 | 42.4 | 17 | 51.5 |
| Urban | 19 | 57.6 | 16 | 48.5 |
| Marital Status | | |
| Married | 33 | 100.0 | 30 | 90.9 |
| Unmarried | | 3 | 9.1 |

Table II(a) shows the mean scores for depression and it's factors on BDI in experimental group a day before institution
TABLE-II(a). BDI scores in experimental (before treatment) and control group

|                              | Experimental group | Control group |
|------------------------------|-------------------|---------------|
|                              | (N=33)            | (N=33)        |
| Mean s.d. Mean s.d. t value  |                   |               |
| Beck's Depression Inventory  |                   |               |
| Depression                   | 42.39 4.64 4.97 2.46 40.31*** |               |
| Guilty depression            | 18.27 2.11 1.85 1.33 39.10*** |               |
| Retarded depression          | 14.21 2.42 2.36 1.62 23.24*** |               |
| Somatic disturbance          | 6.21 1.47 0.73 1.13 16.72*** |               |
| d.f. for all comparisons is 64, ***—p<0.001, **—p<0.01 |               |

of therapy and those of control subjects. Mean scores for depression indicate that experimental group was severely depressed.

TABLE-II(b). Memory scores of experimental (before treatment) and Control Group

|                              | Experimental group | Control group |
|------------------------------|-------------------|---------------|
|                              | (N=33)            | (N=33)        |
| Mean s.d. Mean s.d. t value  |                   |               |
| MQ                           | 77.79 11.70 96.45 7.59 7.57*** |               |
| Mental Control               | 18.0 3.91 21.73 2.01 4.8*** |               |
| Personal & Current information| 3.52 1.26 4.45 0.98 3.30*** |               |
| Orientation                  | 4.76 0.49 5.00 0.00 2.89** |               |
| Mental Control               | 4.64 2.43 6.94 1.36 4.67*** |               |
| Digit forward                | 5.09 0.93 5.33 0.68 1.18 |               |
| Verbal Learning              | 17.52 5.01 23.82 4.21 5.44*** |               |
| Logical memory               | 4.10 1.66 6.94 1.99 6.2*** |               |
| Digit backward               | 3.45 0.8 4.03 0.57 3.34** |               |
| Associate Learning           | 9.97 3.7 12.85 3.02 3.49*** |               |
| Visual Reproduction          | 8.55 2.99 11.85 1.92 3.25*** |               |
| d.f. for all comparisons is 64, ***—p<0.001, **—p<0.01 |               |

Table-II(b) shows that performance of depressions on Memory scale (WMS) was considerably poor in comparison to Control (non-depressed) group as evident from Statistically Significant difference in their MQ scores. The same is true for all the 3 subfunctions and all the items of WMS except digit forward item.

TABLE-III(a). BDI scores in experimental group (before and after treatment)

|                              | Before Treatment | After Treatment |
|------------------------------|-------------------|-----------------|
|                              | (N=33)            | (N=33)          |
| Mean s.d. Mean s.d. t value  |                   |                 |
| BDI Depression               | 42.39 4.64 8.48 3.34 33.57*** |               |
| Guilty depression            | 18.27 2.11 3.39 1.81 35.61*** |               |
| Retarded depression          | 14.21 2.42 4.39 1.84 21.86*** |               |
| Somatic disturbance          | 6.21 1.47 0.36 0.8 26.67*** |               |
| d.f.—32, ***—p<0.001 |               |

(Scores of depression in experimental subjects before institution of therapy and on final assessment on BDI after treatment, indicate substantial recovery from depression. There was highly significant difference between the scores for depression and its factors on two assessments).

Table-III(b) shows the mean scores of experimental group before and after treatment on WMS. Corresponding with the lifting up of depression after clinical recovery, significant increase in MQ and various subfunctions of memory was observed (p<.001). Similarly significant improvement was observed on all the items of W. M. S.

Table-IV shows correlation between scores for depression and its factors, and scores for MQ and 3 subfunctions of memory on WMS in experimental group before treatment. A negative correlation between depression and memory is evident from
TABLE-III(b). Memory scores of experimental group (before and after treatment)

|                   | Before Treatment (N=33) | After Treatment (N=33) |
|-------------------|-------------------------|------------------------|
| **Mean s.d.**     | **Mean s.d.**           | **t value**            |
| **MQ**            | 77.79 11.70            | 94.88 10.57           | 14.32*** |
| Mental Control    | 18.0 3.91              | 21.0 2.98             | 8.37*** |
| Personal and current information | 3.52 1.26    | 4.00 1.21             | 3.94*** |
| Orientation       | 4.76 0.94              | 4.94 0.34             | 2.65*   |
| Mental Control    | 4.64 2.43              | 6.61 1.77             | 8.22*** |
| Digit forward     | 5.09 0.93              | 5.45 0.70             | 3.83*** |
| **Verbal Learning** | **Logical Memory**   | **Digit backward**    | **Associate Learning** |
|                   | 17.52 5.01             | 23.76 11.08          | 11.08*** |
|                   | 4.10 1.66              | 6.67 1.88             | 9.74*** |
|                   | 3.45 0.8               | 3.97 0.57             | 5.97*** |
|                   | 9.97 3.7               | 13.12 2.88            | 7.48*** |
| **Visual Reproduction** | **8.55 2.99**          | **11.18 1.82**        | **6.09*** |
| df—32, *—p<0.05, ***—p<0.001

TABLE-IV. Correlation between scores for depression and its factors, and scores for MQ and its subfunctions (depressed group)

| Depression     | MQ | Mental | Verbal | Visual | Control | Learning | Reproduction |
|----------------|----|--------|--------|--------|---------|----------|--------------|
| Depression     | -0.51** | -0.60*** | -0.46** | -0.19 |
| Guilty Depression | -0.10  | -0.22  | -0.10  | -0.10 |
| Retarded Depression | -0.27 | -0.36** | -0.34  | -0.4 |
| Somatic Disturbance | -0.23 | -0.39** | -0.003 | -0.30 |

*p<0.05; **—p<0.01; ***—p<0.001

It is evident from Table V that change in depression was correlated with memory (MQ) and its subfunctions, but this correlation was significant only with change in mental control subfunction which is also significantly correlated with change in somatic disturbance factor of depression. No significant correlation is observed between changes in guilty depression factor and retarded depression factor with changes in MQ and its various subfunctions.

DISCUSSION

From results of present enquiry it is apparent that primary depression is associated with impairment of memory. Compared with memory scores of control subjects on WMS, performance of depressives was significantly poor (Table II b). Moreover with the lifting up of depression evident from statistically significant change in BDI scores (Table-III a), there was an overall significant improvement in MQ and various subfunctions of memory (Table II b).

Impairment in memory functions in depressives after recovery has been observed by several investigators (Zamora & Kebelbing, 1965; Post, 1966; Holliday et al., 1968), but their observations are not strictly
comparable to those of ours because their studies were designed to achieve an altogether different aim of assessing the effect of ECT on memory functions or comparing the amnesic effect of unilateral and bilateral E.C.T.

There are only few studies in literature primarily aimed at assessing the influence of depression on memory function. Cronholm & Ottosson (1961) compared the memory performance of 42 cases of endogenous depression 1-2 days before 1st and one week after the last EGT. Improvement in immediate reproduction (learning) was seen after clinical recovery. Similar results were obtained by Stromgren (1977) who assessed memory functions of 152 endogenously depressed patients on WMS. Sternberg & Jarvik (1976) also observed improvement in immediate reproduction in patients of endogenous depression after recovery. Results of all these workers are in conformity with our observations reflecting a significant improvement in all sub-function of memory and MQ (Table III b), consequent to lifting up of depression after treatment (Table III a). Cronholm & Ottosson (1961) and Sternberg & Jarvik (1976) also compared performance of depressives with matched controls. Observation of both investigators are similar to ours in finding memory performance of normal controls significantly superior to that of depressives (Table II b). Poor performance of depressives as compared to matched controls can be attributed to the effect of depression on memory functions.

Kendrick and Post (1967) did not find any difference between depressed patients and normal subjects on various memory tests either before institution of therapy or 6 and 12 weeks later. Authors excluded those depressives from their study who scored in brain damaged range on synonym learning test. Of these few could have been severe depressives presenting as pseudo-dementias. Exclusion of such cases would result in some increase in pretreatment mean memory scores of depressives group. Moreover, pseudo-dementias respond readily to treatment and hence this mode of selection would result, to some extent, in elimination of upward revision of memory scores after treatment effecting correlation between depression and memory.

Levy (1968) also has not found gains in memory scores after lifting up of depression by ECT. Rather there was observed a decline in mean scores on WMS by 14 points after recovery. This decline however might be due to the fact, that second memory assessment was done 6 hours after last treatment when dysmnesic effect of ECT might be so pronounced as to mask the memory improvement effect due to lifting up of depression.

Friedman (1964) and Kahn et al. (1975) did not find significant relationship between depression and memory. The reason for theirs coming to such conclusion may be that they studied patients different from those of ours with regard to age group and diagnosis and used different tools for assessment.

In present study, effect of depression and its various factors I—Guilty depression, II—Retarded depression and III—Somatic disturbances (Weckowicz et al., 1967), on memory and its various subfunctions, I—Mental control, II—Verbal learning and III—Visual reproduction was studied with the help of pearson's correlation coefficient. It was observed that in experimental group before treatment (Table IV), Scores on depression were significantly negatively correlated with total scores on memory, mental control and verbal learning. Scores on Visual reproduction were not found to be correlated with total scores on depression significantly. However, various factors of depression do not show significant negative correlation with various subfunctions of memory except mental control subfunction which is most significantly correlated with total scores on depression and its various factors except guilty depression.
Calculation of correlation between changes in depression and those of sub-functions of memory (Table-V) suggest that depression influences various subfunctions of memory adversely and that these subfunctions improve with recovery from depression. Here again, mental control was found to be most sensitive for depression and its factors. Similar results were obtained by Stromgren (1977) who found most significant correlation between mental control and depression along with its factors before treatment and changes in scores of mental control with the change in depression after treatment. However, in our study correlation between other factors of depression and various subfunctions of memory, as observed by Stromgren (1977) were not obtained. This variance may be due to difference in tool used for measuring depression and consequently obtaining different factors of depression.

Significant correlation of depression with mental control indicates that probably depressives are defective in the earliest mechanism of cognitive functions like perception and attention. Fabing (1955) stated that the learning impairment in depression is based on a lowering of awareness. It has been hypothesized that in depression there is basic pathophysiology of attention and alerting mechanisms mediated by the reticular activating system (Zung et al., 1968), thus during sleep, depressives show increased neuronal activity of their arousal system, which produces lighter sleep, more frequent awakenings and greater sensitivity to external stimuli than normal in an equivalent stage of sleep (Zung et al., 1964). Thus learning impairment seen during the day could be based on a decreased level of awareness via decreased neuronal activity of R.A.S.

Retrieval is not an independent phenomenon and is intimately connected with assessment of memory function as a whole. Therefore, its individual role on memory impairment seen in primary depression can only be indirectly inferred. Retrieval demands initiation, on the part of the person, of information—recovery and choosing the necessary system of connections from all the possible alternatives. It is therefore, likely that emotional, motivational and cognitive factors greatly influence retrieval ability. From learned helplessness model of depression (Seligman, 1972; 1973; Miller and Seligman, 1975), it is evident that undermined self-esteem and feeling of helplessness limit the efforts required for memory retrieval. Energy and motivation necessary for retrieval are limited in depressed patients.

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