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ECT and non-memory cognition: A review

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The literature on the effects of electroconvulsive therapy (ECT) on non-memory cognitive functions is reviewed. It is concluded that with early methods of ECT administration (sine wave, high dose), these effects are larger than those of depression. They are less pronounced, and usually do not exceed the effects of depression, when modern methods of ECT administration (brief pulse, moderate or low dose) are used. Following ECT, these functions progressively improve. At one week to seven months after ECT, performance is better than before ECT, probably because of the alleviation of both the effects of depression and of ECT. The time course to full recovery of the non-memory effects resembles that of the recovery of amnesic effects, although the latter are more pronounced. With bilateral ECT, as with right unilateral ECT, there is evidence that right hemisphere effects are more pronounced. The results of this review argue that clinicians should take the non-memory cognitive effects of ECT into account, and patients should be informed of their existence before they sign consent for ECT.

It is well established that electroconvulsive therapy (ECT) transiently affects cognition (Calev, Pass, Shapira, Fink, Tubi & Lerer, 1993; Sackeim, 1992; Squire, 1984). Immediately after ECT, patients experience post-ictal disorientation (Calev, Cohen, Tubi, Nigal, Shapira, Kugelmass & Lerer, 1991*a*) and soft neurological signs such as headaches, confusion and psychomotor slowing (Tubi, Calev, Nigal, Shapira, Fink, Pass, Jandorff & Lerer, 1993). These acute effects usually resolve within hours. During the weeks after ECT, memory dysfunction is observed (Calev, Nigal, Shapira, Tubi, Chazan, Ben-Yehuda, Kugelmass & Lerer, 1991*b*).

This memory dysfunction is characterized as organic amnesia (Calev *et al.*, 1993; Squire, 1984). After ECT, patients experience rapid forgetting of new information (anterograde amnesia). They also have difficulty remembering events occurring prior to ECT (retrograde amnesia). Events occurring earlier in life are better remembered than events which occurred shortly before the treatment (e.g. Squire, 1984). Events which occurred during the treatment course may be permanently forgotten (Calev *et al.*, 1993; Squire, 1984). The disorientation that occurs minutes after ECT has also been described as a memory dysfunction, and, as in retrogade amnesia, ECT patients have better orientation for information acquired earlier in life than for information acquired later (Calev *et al.*, 1991*a*; Daniel, Crovitz & Weiner, 1987). These effects gradually improve and at about six months after treatment, most researchers agree that memory effects are no longer present (Calev *et al.*, 1993).

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These amnesic effects of ECT have been attributed to the fronto-temporal electrode placement typically used, and the lower seizure threshold of medial temporal brain structures associated with memory function (e.g. Sackeim, 1992; Squire, 1984). It has been argued that the flow of electric current through medial temporal brain areas is responsible for the memory dysfunction (e.g. Sackeim, 1992; Squire, 1984).

Whereas the nature of the memory deficits and post-ictal disorientation has been well defined (see Calev *et al.*, 1993; Squire, 1984, 1986 for reviews), data on the effects of ECT on cognitive functions other than memory and orientation (non-memory functions) are conflicting. Some reports do not show such effects (e.g. Squire, 1975; Taylor & Abrams, 1985; Taylor, Kuhlengel & Dean, 1985) while other reports do (e.g. Kahn, Pollack & Fink, 1960; Pascal & Zeaman, 1951). One can speculate that current flow through the temporal and frontal lobes of the brain should affect both memory and non-memory neuropsychological functions in which these areas are involved. If the electrical stimulus is strong, the seizure should generalize to many brain locations and affect other cognitive functions. If stimulus intensity is low, the seizure may not be generalized and cognitive tasks other than memory may be minimally affected. According to this logic one should expect to find adverse non-memory effects of ECT.

The assessment of the cognitive effects of ECT cannot be done without taking into account the cognitive effects of the depressive or the psychopathological state that precedes ECT (Abrams & Taylor, 1985; Calev *et al.*, 1993; Sackeim, Freeman, McElhiney, Coleman, Prudic & Devanand, 1992*a*). Depression is characterized by a generally lower cognitive performance, mainly on tasks less resistant to deterioration (fluid rather than crystallized intellectual tasks), such as attention, memory and visuospatial perception tasks. General intelligence is also affected, the verbal component less affected than the performance component (Sackeim *et al.*, 1992). Therefore, returning to a pre-ECT level of cognitive functioning, after recovery from depression, does not mean that the effects of ECT have recovered. It only means that the effects of ECT are not larger than the effects of depression (Calev *et al.*, 1993). Some studies did not take the effect of pre-ECT depression and mental illness into account. However, when considering the net effect of ECT, in the present review, these effects have to be taken into account.

The present review attempts to find out whether the research done so far supports the existence of a non-memory cognitive deficit after ECT; to determine whether the time course and the relative magnitude of these deficits resemble that described for amnesic effects; and to evaluate the possibility that certain non-memory functions are more adversely affected by ECT than others.

Method

To address these questions, the studies published since 1975 on non-memory cognition and ECT were reviewed, with the help of a Medline computer search. This search included most of the studies published on non-memory ECT cognition. It thus represents the field fairly well. Other studies known to the research team members were also included, again with no pre-selection. The studies reviewed vary with respect to their scientific rigour. Most studies used depressive patients, although psychotic states and additional diagnoses are also represented. Sample sizes are sufficient for statistical analyses

(usually above a minimum of 10 patients per cell), but not all studies meet a criterion of 80 per cent power. Not all studies describe their method and results sections in sufficient detail, even to evaluate rigour or extract size effects information. Despite these problems, all studies reviewed were included, because of the paucity of research in this area.

Earlier reports than 1975 (reviewed by Abrams, 1992; Fink 1979; and Price, 1982) are not the focus of this review because present-day ECT administration involves different electrical waveform and stimulus intensity resulting in less severe cognitive effects. They are, however, briefly reviewed in order to compare present-day cognitive effects with the effects of higher levels of stimulation, without supply of oxygen and ventilation, before 1975.

In this review cognitive effects of ECT are classified into five categories based on the time of their occurrence: (1) acute effects (up to seven hours after ECT); (2) early subacute effects (seven to 72 hours after ECT); (3) middle subacute effects (72 hours to one week after ECT); (4) late subacute effects (one week to seven months after ECT); and (5) long-term effects (seven months or more after ECT). The acute–subacute distinction is now well accepted with respect to memory dysfunction after ECT (e.g. Calev *et al.*, 1993; Sackeim, 1992). The subclassifications within the subacute period, however, were made to estimate accurately the differences in severity of non-memory effects as a function of time.

Results

Acute period (up to seven hours after ECT)

Table 1 shows that studies assessing acute effects of ECT on general intelligence and perceptual function, all having adequate sample sizes, fail to find an effect. Although one of these studies (Kronfol, Des Hamsher, Dirge & Waziri, 1978) made the argument of such an effect, the data presented fail to support it. First, only three out of eight tasks showed an effect, and second, the verbal tasks showing the effect (controlled word association and digit sequence learning) were heavily loaded on memory, which is known to be affected by ECT.

Some well controlled studies support an attention deficit acutely after ECT. Sackeim, Portnoy, Decina, Malitz, Warmflash, Vingiano & Yudofsky (1983) found left visual field inattention (neglect) for items in cancellation tasks. Their patients noticed more syllables and figures in the right than in the left visual field when tested 35 to 50 minutes following ECT. This was observed with both right unilateral and bilateral ECT. In a recent follow-up study, Sackeim, Nobler, Prudic, Devanand, McElhinney, Coleman, Settembino & Madatta (1992*b*) were able to replicate these

Table 1. Acute phase (0–7 hours)

Area	Study	Effect of ECT
General intelligence	Squire, 1975	No change[a]
	Kronfol *et al.*, 1978	No change[a]
Perceptual function	Rossi, Stratta, Nistico, Sabatini, Di Michele & Cassacchia, 1990	No change[a]
Attention	Sackeim *et al.*, 1983	Left-sided inattention
	Sackeim *et al.*, 1992	Left-sided inattention
	MacKenzie *et al.*, 1983	Reduced speed in attention vigilance tasks

[a] No change can be attributed to ECT.

findings with high dose bilateral brief pulse ECT. Right unilateral ECT produced left visual field neglect, even with lower dose stimulus. These findings suggest a right hemisphere effect of ECT.

Another study (MacKenzie, Price, Tucker & Culver, 1985) showed an acute adverse effect of ECT on an attention task assessing vigilance. Between 4.5 and 6.5 hours after ECT a normal practice effect was found for performance speed on three non-memory tasks in control subjects who did not receive ECT (digit symbol from the WAIS, Spokes and letter cancellation). ECT patients showed less improvement in speed than controls on the letter cancellation task (MacKenzie *et al.* 1985). Like Sackeim *et al.*'s (1983) study, this study may implicate an acute attention deficit after

Table 2. Early subacute period (7–72 hours)

Area	Study	Effects of ECT
General intelligence	Squire, 1975	No change[a]
	Calev *et al.*, 1991*b*	No change[a]
	Taylor *et al.*, 1985	No change[a]
	Calev, Gaudino & Fink, in preparation	No change[a]
	Lawson, Inglis, Delva, Rondenburg, Waldron & Letermendia, 1990	Performance IQ drops non-significantly
	McKenna & Pratt, 1983	Improvement (on digit symbol subtest of the WAIS)
Language	Taylor *et al.*, 1985	Results suggestive that verbal fluency[b] is adversely affected
	Lerer, Calev, Tubi, Drexler, Kindler, Lidsky, Schwartz & Shapira, 1995	Verbal fluency[b] adversely affected
	Taylor & Abrams, 1985	No change (on a variety of language tasks)[a]
	Jones, Henderson & Welch, 1988	One of 20 tasks (word fluency, assessing retrieval from semantic memory) affected[b]
Perceptual and visuo-spatial function	Taylor *et al.*, 1985	No change[a]
	Taylor & Abrams, 1985	No change[a]
	Calev *et al.*, 1991*b*	No change[a]
Motor function: manual dexterity	Taylor & Abrams, 1985	No change[a]
Higher cognitive and frontal function	Taylor *et al.*, 1985	No change[a]
	Taylor & Abrams, 1985	No change[a]
	Lawson *et al.*, 1990	No change[a]

[a] No change can be attributed to ECT.
[b] Calev *et al.* (1993) suggest this may be a memory rather than a language problem.

ECT. Alternatively, this latter finding may be interpreted as a memory deficit resulting in an inability to benefit from practice effects, rather than an attention deficit. This leaves Sackeim *et al.*'s (1983, 1992*b*) findings as the main findings showing an attention non-memory deficit after ECT.

Early subacute period (seven to 72 hours after ECT)

Recent studies (after 1975). Table 2 shows that the overwhelming majority of reports fail to find deterioration in non-memory cognitive function at the early subacute phase. Thirteen out of 17 findings reviewed show no change in non-memory cognition after ECT (one of these studies showing a non-significant trend for deterioration). This is a significant proportion ($p = .025$, binomial test, assuming independence among findings). The only area showing deficits (in three studies) was language, and the only task within the language domain was verbal fluency. This task assesses retrieval difficulty perhaps due to psychomotor slowing, and is heavily loaded on memory (Calev *et al.* 1993). Since we know that memory is affected by ECT, this finding cannot be taken to support a language dysfunction *per se* after ECT.

Since the pre-ECT psychopathological state is associated with a lower cognitive performance (e.g. Calev, Korin, Shapira & Lerer, 1986; Gil, Calev, Greenberg, Kugelmass & Lerer, 1990; Sackeim *et al.*, 1992), persistence of these deficits after the resolution of psychopathology (usually depression) after ECT, suggests that the effects of ECT are of comparable magnitude to the effects of depression. Before ECT depressives and other patients are characterized by a generalized cognitive deficit (Calev & Monk, 1982; Cassens, Wolf & Zola, 1988; Chapman & Chapman, 1978; Sackeim *et al.*, 1992) and some more specific deficits (e.g. Calev *et al.*, 1986). Immediately after ECT, many of these deficits persist despite the recovery from depression or mental illness, not because of illness but because of the effects of ECT. At about six months after ECT, patients' cognition improves, probably because both the effects of mental illness and of ECT have recovered (e.g. Abrams & Taylor, 1985; Calev *et al.*, 1993).

Early studies (before 1975). In order to find out whether indeed ECT has an adverse effect on non-memory cognition, six older ECT studies using sine wave ECT, usually at very high doses of electrical stimulation, were sampled from formerly written reviews, based on the immediacy of availability of the reports. In four out of six of these studies, deficits were observed in the early subacute stage (one day after ECT), as compared with pre-treatment. Kahn *et al.* (1960) found an increase in the number of errors on an embedded figures visuospatial task. Fink, Kahn, Karp, Pollack, Green, Alan & Lefkowits (1961) reported a decline in measures of intelligence such as object assembly and on perceptual tasks. Perceptual-motor and frontal-lobe deficits were reported by McAndrew, Berkey & Matthews (1967) including finger tapping speed, maze time, pegboard time and the Category Test. Qualitative speech problems following ECT have also been reported with this type of treatment (e.g. Kahn, Fink & Weinstein, 1956). However, Spreche (1963) reported no major adverse effects of ECT subacutely (48 to 72 hours) on measures of intelligence, visuospatial

perception and motor speed. Similarly Scanlon & Mathas's (1966) data suggest no effect on measures of motor speed (Trail-Making Test) and intelligence. Nevertheless, a substantial number of the early studies using high dose electrical stimulation suggest impairments when comparing the subacute state to the pre-ECT state. These changes have also been observed with convulsive medication (e.g. Pollack, Kahn, Karp & Fink, 1962).

This suggests (see summary in Table 3) that the effects of ECT on perceptual, language and other cognitive functions may be larger than those of depression if stimulus intensity is high. Modern ECT techniques usually employ brief-pulse, moderately suprathreshold stimulation (e.g. 150 per cent of the energy needed to elicit a 20 second first seizure, measured by the cuff method) and age dosing (which gives on average double the energy used with suprathreshold stimulation). Many times unilateral (usually right) ECT is given. Under these conditions the deficits caused by ECT are not larger than those observed during the pre-treatment mentally ill state.

Table 3. Early subacute period (7–72 hours): Early studies (before 1975)

Area	Study	Effect of ECT
Higher executive, motor and perceptual functions	Kahn *et al.*, 1960	Adverse effect (visual-perception)
	Fink *et al.*, 1961	Adverse effect (visual-perception)
	McAndrew *et al.*, 1967	Adverse effect (executive function, motor speed)
	Spreche, 1963	No change[a]
	Scanlon & Mathas, 1966	No change[a]
Language	Kahn, Fink & Weinstein, 1956	Adverse effect (speech)

[a] No change can be attributed to ECT.

Table 4. Middle subacute period (72 hours to one week)

Area	Study	Effect of ECT
General intelligence	Weeks *et al.*, 1980	No change[a]
	Wilkinson & Anderson, 1993	Improvement (mini-mental-state examination)
Language	Weeks *et al.*, 1980	Improvement (sentence repetition better than base-line, in depression)
Perceptual function	McKenna & Pratt, 1983	Improvement
	Weeks *et al.*, 1980	Improvement
Attention and frontal function	Weeks *et al.*, 1980	Improvement (in choice reaction time but no other tasks)

[a] No change can be attributed to ECT.

Middle subacute period (72 hours to one week after treatment)

There only are a few findings on non-memory cognitive function at the middle subacute period (see Table 4). They show that this period is characterized by performance which is similar to or better than that of the pre-ECT (depressed or otherwise mentally disturbed) state.

Late subacute period (one week to seven months after ECT)

Table 5 shows that 12 out of 16 findings were of improvement or improvement trend at the late subacute period, starting one week after ECT. This is not an accidental proportion ($p = .038$, binomial test, assuming independence among findings). The most specific finding reported, namely the restoration of right ear dominance presumed lost during depression (Williams, Iacano, Remick & Greenwood, 1990) requires replication. Weeks, Freeman & Kendell's (1980) study is of great importance since they used a normal control group, and found that, at this stage of the subacute period, the qualitative differences between ECT patients who recover from

Table 5. Late subacute period (one week to 7 months)

Area	Study	Effect of ECT
General intelligence	Small, Milstein, Miller, Malloy & Small, 1986	Improvement
	Malloy, Small, Miller, Milstein & Stout, 1982	Improvement
	Lawson *et al.*, 1990	Improvement
	Weeks *et al.*, 1980	No change[a]
Language	Weeks *et al.*, 1980	No change[a]
Perceptual function	Malloy *et al.*, 1982	Improvement (trend)
	Small *et al.*, 1986	Improvement
	Williams *et al.*, 1990	Improvement (normalizing auditory discrimination abnormality attributed to depression)
	O'Connor, Colter & Shaw, 1984	Improvement (less field dependence relative to depression)
Motor function: manual dexterity	Malloy *et al.*, 1982	Improvement (trend)
	Small *et al.*, 1986	Improvement (trend)
	Weeks *et al.*, 1980	Improvement, i.e. normal performance
Attention and frontal function	Malloy *et al.*, 1982	Improvement
	Small *et al.*, 1986	No change (Category Test)[a]
	Weeks *et al.*, 1980	Improvement (in mental shifts test but not in vigilance)
	Shellenberger, Miller, Small, Milstein & Stout, 1981	No change[a]

[a] No change can be attributed to ECT.

depression and normal controls disappear. These, however, do not seem to occur in the first two or three weeks but seem to be present somewhat later. Replications in well-controlled studies, using normal control groups, are of importance in order to determine if and when non-memory neuropsychological function normalizes after ECT.

Long-term effects

Generally, no lasting effects of ECT on non-memory cognitive functions beyond the subacute period have been found. Abrams & Taylor (1985) assessed ECT patients before ECT, after the sixth treatment, one month, six months and one to two years later, using the same tasks that Taylor & Abrams (1985) used to assess subacute effects. They used a global impairment index and had a control group not receiving ECT. Patients' performance continued to improve gradually up to the last assessment (one to two years after ECT). Beginning with 30 days after treatment, no significant impairment was found in ECT patients when compared to controls. Stoudemire, Hill, Morris, Martino-Salzman & Lewinson (1993) used the Mattis Dementia-Rating Scale and found an improvement in global cognitive functioning in depressed elderly patients who were somewhat impaired cognitively, four years after ECT. Stoudemire, Hill, Morris, Martino-Salzman, Markwalter & Lewinson (1991) reported similar improvement at six months' follow-up. Pettinati & Bonner (1984), however, found an impairment in frontal lobe function, as assessed by slowness on the Trail-Making Test, which requires mental shifts (frontal function). This report, however, cannot be taken as substantial evidence for an impairment when contrasted to the bulk of reports showing no deficit in cognition even at the late subacute period and reports of lack of cognitive impairment following hundreds of ECT treatments over the life-span (e.g. Devanand, Verma, Tirnmalasetti & Sackeim, 1991) and also long-term follow-up studies (e.g. Abrams & Taylor, 1985; Weeks *et al.*, 1980).

Discussion

This review addressed three questions. *The first question* was whether ECT affects non-memory cognitive function; the answer seems affirmative. Despite recovery from depression, there is no improvement in cognition at the early subacute phase after the ECT series, suggestive of a general non-memory cognitive deficit caused by ECT, which appears to be of a comparable magnitude to that caused by severe depression or other psychopathology. However, there are four clarifications to be made: (1) the effects on non-memory function are less severe than those on memory function; (2) the effect of ECT rarely exceeds the effects of depression or other pre-ECT pathology, when using modern ECT techniques (brief-pulse, moderate stimulation); (3) studies using older methods of ECT administration (sine wave, high dose) show more severe effects than those produced by depression or other psychopathology; (4) the acute effect of ECT on attentional tasks appears to be larger than that caused by depression (and of distinct characteristics), even when using present-day ECT administration.

The second question was whether the time course for recovery of these non-memory deficits is similar to the time course for recovery of memory function after ECT. The

answer seems affirmative. As time progresses after ECT, there are more reports of improved cognitive function. *In the earliest acute stage* deficits have been observed, relative to the depressive pre-ECT stage, even with the present-day ECT administration methods. *In the early subacute period* there seems to be some improvement, and cognitive performance resembles that observed prior to ECT (when using present-day ECT administration); performance is poorer than that observed during depression only when high dose and/or sine wave stimuli are used. In *the middle subacute period* an improvement is reported relative to the pre-ECT depressive (or other psychopathological) state, and reports of such improvement are frequent *in the late subacute period.*

The third question concerns the tasks and brain locations that are most affected by ECT. One can only offer a partial answer to this question. The specific left hemisphere neglect reported by Sackeim *et al.* (e.g. 1983, 1992) in the acute phase is suggestive of a right hemisphere effect of both bilateral and unilateral ECT. This finding, however, needs replication by other research groups. Another finding (which also needs replication) is the return of right ear advantage in dichotic listening performance after ECT (Williams *et al.*, 1990). Taken together these two studies suggest a lateralized effect of both bilateral and right unilateral ECT, perhaps due to the postulated right hemispheric involvement in depression.

When comparing frontal lobe (attention shift) functions and parietal (largely perceptual) functions, the results are inconclusive. The research fails to show clearly more pronounced effects on frontal than parietal tasks, as could be expected because of the fronto-temporal electrode placement. It can be speculated that since both these areas are less likely to seize than the medial temporal areas causing amnesia, their functioning is less affected, resulting in no major difference between these two areas.

This review suggests that clinicians and patients should be aware of non-memory effects of ECT as much as they are aware of memory effects. The non-memory effects are substantial, although not larger than the effects of depression. Patients usually prefer to tolerate the adverse memory effects of ECT than to tolerate the effects of depression. However, having non-memory cognitive problems means that patients are not going to function well on more tasks than they anticipate. Yet, ECT is the most effective treatment of depression and the response rate is over 80 per cent (e.g. Abrams, 1992). Patients who experience severe intractable depression value its effects. It is conceivable that most patients who agreed to have ECT will still agree to it, if informed of the extent of non-memory cognitive side-effects. However, patients should be able to know all the facts before making a decision to be treated. The consent forms used by clinicians, including the most comprehensive form recommended by the ECT American Psychiatric Association Task Force, do not mention that such non-memory cognitive effects are caused by ECT. Patients need not be excessively worried by these effects, but they should know and consent to have them, when considering ECT as an option. One hopes this review will help in clarifying this issue to both clinicians and patients.

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