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Changes in Psychiatric Symptoms Related to EEG and Cerebral Blood Flow Following Electroconvulsive Therapy in Depression

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Summary. Changes in psychiatric symptoms following electroconvulsive therapy (ECT) were related to alterations in global EEG and cerebral blood flow (CBF) in 21 in-patients suffering from depression. They were examined by clinical ratings, EEG, and CBF immediately before and 1 to 3 h after treatments during an ECT series and at follow-up. Four symptom clusters from a factor analysis of symptoms in depression, representing different dimensions of emotion, cognition, and psychomotor retardation, were used for clinical description. The changes in the separate symptom clusters showed different patterns and also different correlations with neurophysiological (EEG and CBF) changes during the course of serial ECT. Furthermore, acute clinical and neurophysiological effects following single ECT's were found to be different from non-acute changes, building up during the treatment course. Acute relief in symptoms of anxiety, depressed mood, and psychomotor retardation correlated with an acute slowing of the EEG. Regarding non-acute effects a reversed relationship was found, i.e. improvement in symptoms of depressed mood and psychomotor retardation was related to less EEG slowing. As opposed to the acute clinical changes, the non-acute changes, found following the first two or three treatments of a series, contained predictive information about the individual clinical outcome of the patients.

Key words: ECT – EEG – Cerebral blood flow – Factor analysis – Depression

Introduction

In the study of depressive disorders, EEG has frequently been used as a neurophysiological correlate to effects of treatment. Several studies have shown a lack of relationship between pre-treatment clinical psychopathology and global EEG (Kennard and Willner 1948; Nilsson and Smith 1965). However, attempts have been made to relate symtoms of depression to hemispheric and regional quantified EEG findings (d'Elia and Perris 1973; Monakhov et al. 1979). There is a general agreement about the fact that serial electroconvulsive therapy (ECT) causes a slowing of the EEG in most patients (Meyer-Mickeleit 1949; Chusid and Pacella 1952; Fink and Kahn 1957; Ottosson 1960; Nilsson and Smith 1962). There are, however, marked inter-individual differences in the amount, rate, and persistence of the EEG slowing (Klotz 1955; Green 1957; Weiner 1980, 1981). These EEG differences following ECT have been related to the pre-treatment EEG, the individual reactivity to seizures, ECT method, the number of treatments, and the duration and frequency of seizures (Kennard and Willner 1948; Chusid and Pacella 1952; Green 1957, 1960; Stein et al. 1968; d'Elia 1970; Volavka et al. 1972; Robin and Tissera 1982). Nilsson and Smith (1965) studied the relation between clinical and EEG changes from before to after serial ECT using an automatic optical analysis of EEG frequency (Krakau 1951). They found indications of a positive correlation between EEG slowing and clinical improvement. Such a correlation had also been reported by Fink and Kahn (1957). In contrast, Volavka (1974) and Kurland et al. (1976) reported smaller amounts of slow waves in the clinically most improved compared to the less improved patients suffering from depression.

There are few reports concerning the measurements of regional cerebral blood flow (rCBF) during ECT in depression. Since the first report (Silfverskiöld et al. 1979) Prohovnik and coworkers (1986) have found a lower pre-treatment rCBF level in patients responding favourably to ECT compared to non-responders but nothing has been reported regarding possible relations between clinical and rCBF changes during ECT.

The aim of the present study was to analyse the relation between changes in clinical symptoms and changes in global EEG and global CBF in depressed patients during and after serial ECT. The material and design have been presented in detail elsewhere (Silfverskiöld et al. 1987). Details of ECT effects on EEG have also been described separately (Rosén and Silfverskiöld 1987). This study is part of an investigation on affective disorders and treatment effects following ECT from which preliminary findings have been reported (Silfverskiöld et al. 1979, 1983, 1984, 1986).

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Table 1. A. 28 Observed symptoms from a rating scale for affective symptoms (RAS scale). **B** Five factors from a factor analysis of ratings in patients with affective and anxiety disorders

A	B ($n = 117$)	Factor loading
Manic-Depressive Symptoms	*1. Anxiety-Agitation VP 4.7*	
1. Awareness of illness	Anxious facial expression	0.87
2. Lack of judgement – depressive type	Tense, jerky movements	0.86
3. Lack of judgement – manic type	Global degree of anxiety	0.82
4. Depressed peri-orbital expression	Anxious roving gaze	0.80
5. Manic gaze	Tense posture	0.67
6. Speech tempo – decreased	Restlessness	0.56
7. Speech tempo – increased	Vegetative symptoms	0.46
8. Spontaneity of movement – decreased	*2. Mania VP 4.1*	
9. Restlessness	Global degree of mania	0.91
10. Motor tempo – decreased	Lack of judgement	0.89
11. Motor tempo – increased	Awareness of illness	0.87
12. Introverted aggressiveness	Manic gaze	0.74
13. Extraverted aggressiveness	Speech tempo – increased	0.59
14. Global degree of depression	Motor tempo – increased	0.51
15. Global degree of mania	*3. Cognitive Dysfunction VP 4.0*	
	Spatial disorientation	0.90
Symptoms of Cognitive Dysfunction	Temporal disorientation	0.83
16. Temporal disorientation	Impaired ability to understand questions	0.83
17. Spatial disorientation	Dysmnesia for persons, names	0.77
18. Dysmnesia for persons, names	Incoherence	0.71
19. Incoherence	Impaired ability to give adequate answers	0.66
20. Impaired ability to understand questions	*4. Depression-Retardation VP 3.9*	
21. Impaired ability to give adequate answers	Motor tempo – reduced	0.89
	Spontaneity of movement – decreased	0.88
Symptoms of Anxiety	Depressed peri-orbital expression	0.84
22. Anxious roving gaze	Speech tempo – reduced	0.82
23. Anxious facial expression	Global degree of depression	0.65
24. Tense posture	*5. Self-depreciation VP 1.9*	
25. Tense, jerky movements	Introverted aggressiveness	0.87
26. Tense strained voice	Self-depreciation	0.80
27. Vegetative symptoms	Global degree of depression	0.44
28. Global degree of anxiety		

Patients and Methods

Patients

In total 21 patients (7 men, 14 women) referred for ECT were studied by clinical psychiatric ratings, EEG, and rCBF. Of these patients, 13 (4 men and 9 women) with a mean age of 63.3 ± 11.6 years received non-dominant unilateral (UL) ECT, and 10 patients (4 men and 6 women) with a mean age of 64.7 ± 13.2 years received bilateral (BL) ECT. The data presented are based on 13 series of UL and 11 series of BL ECT in 21 patients. For further details concerning the material (diagnoses, methods, and results), see Silfverskiöld et al. (1987).

Methods

Design. The patients were examined with clinical ratings, rCBF, and EEG immediately before and 1–3 h after the first ECT (ECT 1), the third or fourth ECT (ECT 3–4), and the fifth to seventh ECT (ECT 5–7), at follow-up within 3 months after the last ECT (clinical ratings only), and at follow-up 4–9 months after the ECT series. When the patients were re-examined following a single treatment, they were fully awake and oriented.

Clinical ratings. The clinical psychiatric evaluation was made by a psychiatrist (P.S.) during a semi-structured interview using specially designed Rating scales for Affective Symptoms (RAS scale) The scale included 28 observed symptoms (Table 1) and was developed from earlier work on affective disorders (Nilsson 1960; Nilsson and Smith 1962; Eberhard et al. 1965). The symptoms were scored in two dimensions: frequency (7 steps: 0 = never; 1 = sometimes; 2 = often; 3 = constantly present during the interview and three intermediate rating steps) and intensity (4 steps: 0 = no/normal; 1 = slight; 2 = moderate; 3 = high during the interview) and the product of these two scores was used. A factor analysis was made on ratings from 117 psychiatric in-patients suffering from affective

or anxiety disorders (Silfverskiöld et al. 1986). The factor analysis produced five symptom clusters (factors) with VP exceeding 1.5, explaining 68% of the total variance. VP is the variance explained by the factor. Four of these factors: depression-psychomotor retardation (depression-retardation), self-depreciation, anxiety-agitation, and cognitive dysfunction, were utilized here for clinical description and for correlation with neurophysiological variables. Standardized factor scores (McNemar 1959) were used. The symptom profiles with factor loadings above 0.40 are presented in Table 1. The fifth factor containing symptoms of the manic syndrome was not used in the present analysis due to the small clinical changes and variance during the ECT series.

The depression-retardation factor mainly reflects the psychomotor symptoms of the melancholic syndrome. "Motor tempo – decreased" means a slowing of movements while "spontaneity of movements – decreased" refers to less amount of movements in the patients. "Depressed peri-orbital expression" is characterized by a heavy, depressive, more or less immobile fixed expression round the eyes. This special mimical symptom which is called "eye retardation" (Silfverskiöld 1958), has been found to have special diagnostic value and may exist with or without other symptoms of psychomotor retardation. The rater's estimation of "global degree of depression" was based on the report of information and on observations during the semi-structured interview.

The self-depreciation factor: "introverted aggressiveness" means excessive self-criticism and self-accusation, feelings and thoughts of satiety with life and suicidal thoughts.

The anxiety-agitation factor: "anxious facial expression" means unmodulated features with facial expressions changing suddenly and more or less uncontrollably. "Anxious roving gaze" means that the eyes are alert, observing and the gaze restless, rovingly anxious.

The cognitive dysfunction factor concerns symptoms of cognitive impairment. "Spatial disorientation" refers to a difficulty or inability to identify room, ward, clinic or hospital area; "temporal disorientation" means a failing awareness of time including hour, day and date; "dysmnesia for persons, names" means difficulty in recognition of a known staff-member and his/her name; "incoherence" is characterized by the patient's difficulty in focusing attention, jumping from one topic to another and showing an increased distractibility by irrelevant stimuli; "impaired ability to give adequate answers" means that even if the patient gives the impression of understanding the question, he is unable to answer adequately.

EEG. A quantitative frequency analysis of the EEG from four bipolar channels was made on each occasion. From the total frequency range of 0.5–25 Hz the EEG power within the 0.5–8 Hz range related to the patient's value at the pre-ECT examination (low frequency power index, LFPI) and the frequency of the dominant EEG activity (dominant frequency, DF) were used.

rCBF. rCBF was measured during rest in 16 regions of each hemisphere by the ^{133}Xe inhalation technique. The initial slope index (ISI) was used corrected for pCO_2. Only mean flow values of both hemispheres will be presented in this communication.

ECT. Conventional ECT equipment (Siemens Convulsator 622) and procedure were used including either right non-dominant UL or BL ECT techniques during brief narcosis.

Statistics. The paired *t*-test was used to evaluate clinical and physiological changes during the ECT series. The Spearman rank correlation coefficient test was applied to evaluate correlations between changes of various variables during the ECT series. The one-way ANOVA test was used to evaluate differences between separate groups of subjects.

Results

The clinical effects of ECT in our material of depressed patients were favourable. Out of 24 ECT series only 4 (2 UL and 2 BL) series did not give a satisfactory therapeutic effect. One of the two UL patients with incomplete recovery after 11 ECT, responded well to 3 additional BL treatments. At pre-ECT 3–4 the BL group showed a higher ($P < 0.05$) cognitive dysfunction score compared to the UL group, but since the groups in all other aspects had similar results they were pooled. The mean number of treatments was similar in the two groups (UL group 7.7 ± 2.4, range 5–14; BL group 7.8 ± 2.3, range 6–14). Before treatment and at follow-up, 4 to 9 months after completion of the ECT series, the patients by and large showed a normal global EEG and CBF. One patient had a relapse of depression during the follow-up period.

The results will be presented in three parts: (1) acute effects of single ECT's calculated from before to after treatment, (2) non-acute effects during the ECT series measured from before the first treatment up to pre-ECT 3–4 and pre-ECT 5–7, (3) clinical outcome of the ECT series related to acute and non-acute clinical and neurophysiological changes during the ECT series. The clinical outcome was calculated from before the first ECT to follow-up within 3 months (usually within 2 weeks) after the last treatment. There is occasional variation in the number of patients due to missing data.

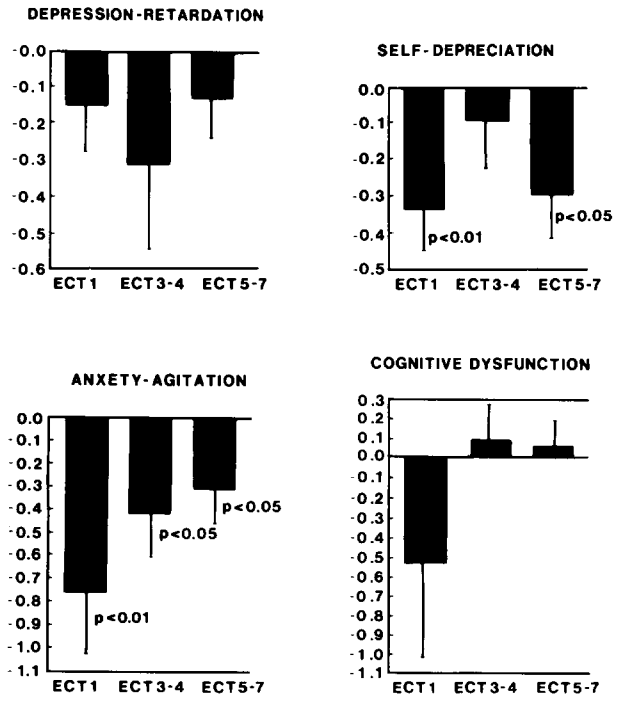

Fig. 1. Acute changes in factor scores during ECT in depression. The mean differences in the factor scores (post- minus pre-ECT values) were calculated. A standardized normal distribution scale, standard errors of the mean (SEM) and *P* values (paired *t*-test) were used. ECT 1 $n = 21$; ECT 3–4 $n = 17$; ECT 5–7 $n = 19$

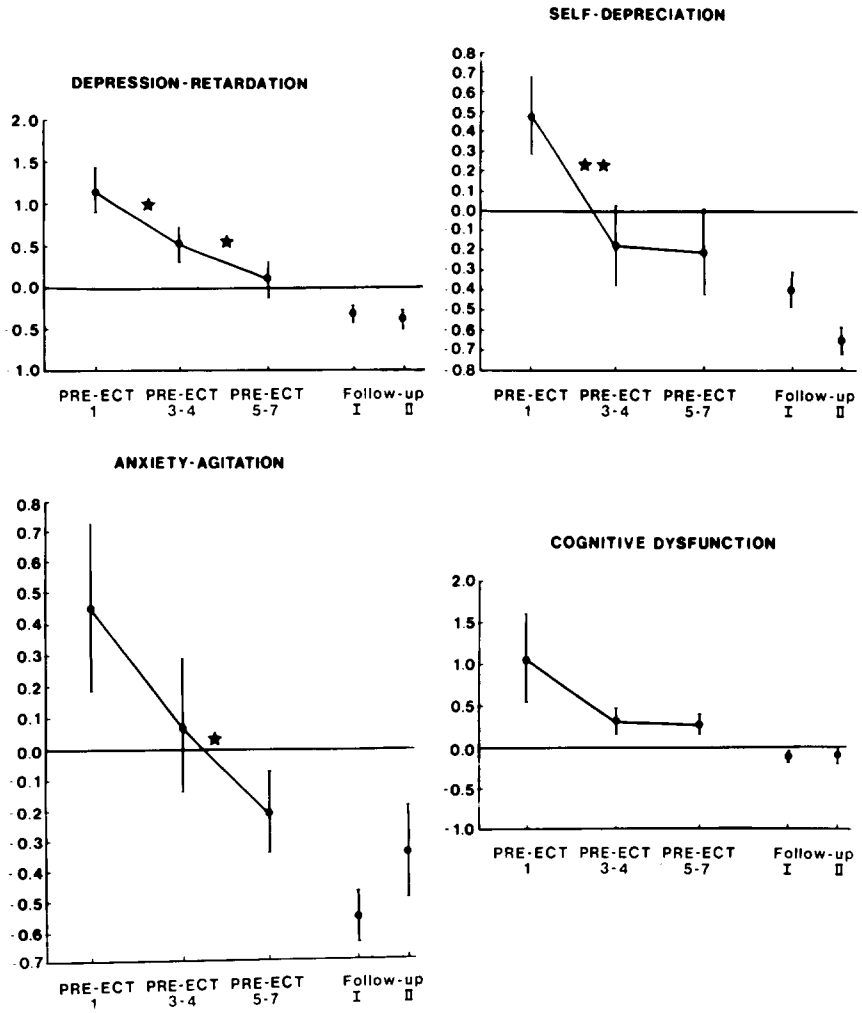

Fig. 2. Pre-ECT values and non-acute changes in clinical factor scores during serial ECT in depression and at follow-up. Means, standard errors of the mean (SEM) and *P* values (paired *t*-test) are given. The change from pre-ECT 1 to pre-ECT 5–7 was significant on the 5% to 0.1% level for the depression-retardation, self-depreciation and anxiety-agitation factor scores. The change from pre-ECT 1 to the follow-up examinations was significant on the 1% to 0.1% level for these three factor scores. ECT 1, ECT 3–4 and ECT 5–7 $n = 22$; follow-up 0–3 months $n = 23$; follow-up 4–9 months $n = 17$. * = $P < 0.05$ (two-tailed); ** $P < 0.01$ (two-tailed)

Table 2. Acute effects of electroconvulsive therapy (ECT) in depression. Correlations between changes (post- minus pre-ECT values) in clinical factor scores and changes in EEG (LFPI, DF) and cerebral blood flow (CBF) (ISI)

	ECT 1 ($n = 20$)			ECT 3–4 ($n = 16$)			ECT 5–7 ($n = 18$)		
	LFPI	DF	ISI	LFPI	DF	ISI	LFPI	DF	ISI
Depression-retardation	0.01	−0.07	0.34	−0.28	0.42	0.29	−0.57*	0.38	0.19
Self-depreciation	0.18	−0.07	0.15	0.04	0.04	−0.28	−0.27	−0.22	0.50*
Anxiety-agitation	−0.48*	−0.06	−0.35	−0.32	0.26	0.42	0.15	−0.53*	0.15
Cognitive dysfunction	0.04	0.14	−0.16	−0.31	−0.36	0.44	0.38	0.09	0.00

LFPI = low frequency power index; DF = dominant frequency; ISI = initial slope index
* = $P < 0.05$ (two-tailed)

Acute Effects of Single ECT's

The acute effects of ECT on the clinical factor scores are presented in Fig. 1. The rated score of the depression-retardation factor decreased on all three occasions. The most marked although insignificant acute effect was found at ECT 3–4. In contrast, the self-depreciation factor score decreased significantly following ECT 1 and ECT 5–7. The changes in the anxiety-agitation score were significant at all stages of the series and most marked at the first ECT. The score of the cognitive dysfunction factor showed an insignificant decrease following the first ECT, and minute changes later in the series.

The acute changes in the clinical factor scores, EEG and CBF were inter-correlated as shown in Table 2. The improvement in the depression-retardation factor was significantly associated with an augmentation of slow waves in the EEG during the latter part of the ECT series. The change in the self-depreciation score and change in ISI were significantly related at ECT 5–7. No such correlation, however, was seen earlier during the series. The anxiety-agitation score showed a significant negative correlation with LFPI changes following ECT 1 suggesting that the initial clinical improvement was associated with an increase in slow waves in the EEG. A significant negative correlation appeared between the anxiety-agitation factor

Table 3. Correlation between clinical outcome of serial ECT and acute effects of single ECT's on EEG (LFPI, DF) and CBF (ISI)

	ECT 1 ($n = 22$)			ECT 3–4 ($n = 20$)			ECT 5–7 ($n = 21$)		
	LFPI	DF	ISI	LFPI	DF	ISI	LFPI	DF	ISI
Depression-retardation	0.27	0.11	0.54**	0.18	0.12	0.16	0.44*	−0.24	−0.11
Self-depreciation	0.16	0.34	0.44*	−0.08	0.01	0.12	0.41	0.10	−0.03
Anxiety-agitation	−0.08	0.05	−0.02	−0.72***	0.34	0.32	−0.13	−0.28	−0.07
Cognitive dysfunction	0.03	0.19	0.02	−0.55*	0.38	0.22	−0.21	−0.07	0.02

LFPI = low frequency power index; DF = dominant frequency; ISI = initial slope index
* = $P < 0.05$ (two-tailed); ** = $P < 0.01$ (two-tailed); *** = $P < 0.001$ (two-tailed)

Table 4. Correlations between clinical outcome of serial ECT and non-acute effects of ECT (pre-ECT 3–4 and 5–7 minus pre-ECT 1) on EEG (LPFI, DF) and CBF (ISI)

	LFPI		DF		ISI	
	Pre-ECT 3–4 − Pre-ECT 1 $n = 19$	Pre-ECT 5–7 − Pre-ECT 1 $n = 22$	Pre-ECT 3–4 − Pre-ECT 1 $n = 19$	Pre-ECT 5–7 − Pre-ECT 1 $n = 22$	Pre-ECT 3–4 − Pre-ECT 1 $n = 19$	Pre-ECT 5–7 − Pre-ECT 1 $n = 22$
Depression-retardation	0.45*	0.36	−0.13	−0.21	0.24	0.50*
Self-depreciation	0.04	0.05	0.00	−0.02	0.33	0.35
Anxiety-agitation	−0.12	−0.29	0.09	0.22	0.38	0.28
Cognitive dysfunction	−0.14	−0.24	0.06	0.20	0.29	0.20

* = $P < 0.05$ (two-tailed)

score and DF at ECT 5–7, indicating that late improvement was related to a lower decrease of DF. There were no significant correlations between acute changes in the cognitive dysfunction factor and changes in the neurophysiological variables (including seizure duration).

Changes in the depression-retardation score showed a significant negative correlation with seizure duration at the first ECT ($r = -0.47$ $P < 0.05$) indicating that the longer the epileptic seizures, the better the therapeutic effect.

Non-acute Effects During the ECT Series

The clinical factor scores are presented in Fig. 2 using the pre-ECT values and the values at the two follow-up examinations. Depression-retardation and anxiety-agitation scores showed successive decreases during the ECT series and at follow-up. The decreases during ECT were significant except for anxiety-agitation between pre-ECT 1 to pre-ECT 3–4 ($P < 0.06$). The self-depreciation factor score showed an initial highly significant decrease followed by a rather stable level later in the ECT series. The scores of the cognitive dysfunction factor changed in a similar way, but the differences did not reach a significant level. Regarding the non-acute clinical and neurophysiological variables a significant positive correlation ($r = 0.44$ $P < 0.05$) was found between changes in the depression-retardation scores and changes in LFPI between pre-ECT 1 and pre-ECT 5–7. Thus, more pronounced clinical improvement was coupled to a smaller increase in slow waves in the EEG. A similar relation ($r = 0.43$ NS) was found also for the pre-ECT 1 to pre-ECT 3–4 interval. This tendency was supported by negative although insignificant correlations between changes in the depression-retardation scores and DF changes at both ECT intervals ($r = -0.34$ NS; $r = -0.29$ NS), i.e. the more clinical improvement the smaller decrease in DF. There were no significant correlations between changes in CBF level and factor scores. No consistent significant correlations were found between the accumulated seizure duration and clinical effects.

Clinical Outcome of Serial ECT Related to Acute and Non-Acute Clinical and Neurophysiological Changes During ECT

The relation between clinical outcome and acute neurophysiological changes is shown in Table 3. Improvement in the depression-retardation and self-depreciation factors correlated significantly and positively with flow reduction at ECT 1 and negatively with EEG slowing at ECT 5–7. Improvement in the anxiety-agitation and cognitive dysfunction factors correlated significantly with EEG slowing at ECT 3–4.

Regarding relations between clinical outcome and acute clinical effects of single treatments a consistent correlation was found only for anxiety-agitation. This correlation reached a significant level at ECT 1 ($r = 0.45$ $P < 0.05$) with a similar tendency regarding cognitive dysfunction.

The relationship between clinical outcome and non-acute neurophysiological changes are shown in Table 4. Only improvement in symptoms of depression-retardation correlated significantly with the neurophysiological changes, namely with less EEG slowing from ECT 1 to ECT 3–4.

As expected the correlations between clinical outcome in a factor and a non-acute improvement of this factor during the series showed significant correlations for all factors at both intervals (pre-ECT 3–4 and 5–7 minus pre-ECT 1: depression-retardation $r = 0.67$ and 0.71 $P < 0.001$; self-depreciation $r = 0.69$ and 0.68 $P < 0.001$; anxiety-agitation $r = 0.46$ $P < 0.05$ and 0.55 $P < 0.01$; cognitive dysfunction $r = 0.67$ and 0.82 $P < 0.001$). Clinical outcome did not show any significant correlation with the total amount of seizure duration of the whole ECT series.

Discussion

When analysing the effects of ECT on psychopathology in depressed patients, two different effects could be discerned: one acute, describing changes following single ECT and one more slowly developing effect during the ECT series which also remained during the follow-up period. The four symptom clusters produced by the factor analysis seemed to be well suited for the clinical description of depressive states. The factors displayed two dimensions of emotions in depression, namely depressed mood and anxiety, in a similar way as has been found in other factor analysis studies (Mullaney 1984). The two factors, depression-retardation and self-depreciation, enabled a further separation of the depressive symptomatology into two aspects – one psychomotor and the other linked to the patient's report of thoughts and feelings. The construct validity of the factors was indicated by the high factor loadings (Table 2) and the similarity with the dimensions of depressive symptomatology, such as retardation and tension, previously described by Nilsson (Nilsson 1960; Nilsson and Smith 1962). The independent changes in factor scores and their independent correlations with neurophysiological parameters following ECT may reflect the meaningfulness of the factor structure. The changes in factor scores presumably indicate more or less specific effects of ECT on the different symptom clusters.

The acute decreases in the depression-retardation factor score did not reach significant levels or correlations to clinical outcome (Fig. 1). In contrast, the non-acute improvement during the ECT series was significant (Fig. 2), and correlated significantly with clinical outcome. Thus the non-acute improvement in the depression-retardation symptoms strongly outweighed the acute relief of the symptomatology. Acute clinical changes tended to correlate with a more pronounced EEG slowing (Table 2), while non-acute improvement and clinical outcome were associated with less non-acute EEG slowing (Table 4). The relation between CBF level and depression-retardation was indicated by the correlations between the clinical outcome of this factor and the acute decrease in ISI following ECT 1 (Table 3), and the non-acute decrease from pre-ECT 1 to pre-ECT 5–7 (Table 4) as previously reported (Silfverskiöld et al. 1986).

The acute reductions in the self-depreciation factor score reached significant levels at the beginning (ECT 1) and at the end (ECT 5–7) of the ECT series and a significant non-acute change was found between ECT 1 and ECT 3–4 (Figs. 1, 2). Although the acute symptom relief was significant, similar to the depression-retardation factor, it showed no significant correlation with the clinical outcome. This was in contrast to the strong correlations between the non-acute changes already seen at the pre-ECT 3–4 examination and clinical outcome. The analogy with the previous factor was also indicated by the very similar correlations between the clinical outcome and the CBF decrease following ECT 1 and the EEG slowing following ECT 5–7 (Table 3). The correlation between flow decrease and clinical outcome of the self-depreciation factor was, however, not as strong as that found regarding the depression-retardation factor (Table 4).

The scores of the anxiety-agitation factor showed significant acute changes following each ECT, with continuous non-acute decreases during the ECT series similar to those of the depression-retardation factor (Figs. 1, 2). Thus, a similar gradual improvement in acute clinical effects was found in factors with different magnitude. It might be noted that the acute improvement in the anxiety-agitation factor showed a correlation with acute EEG slowing at the beginning of the ECT series in contrast to the depression-retardation factor which showed a corresponding correlation towards the end of the ECT series (Table 2). Clinical outcome of the anxiety-agitation factor had a relationship with acute improvements of this symptomatology and with EEG slowing following ECT 3–4 in contrast to the depression-retardation and self-depreciation factors (Table 3). Anxiety-agitation was the only factor in which an acute clinical change (at ECT 1) showed a significant correlation with clinical outcome.

An acute major improvement regarding symptoms of cognitive dysfunction was seen only following the first ECT (Fig. 1) and there were no indications of negative ECT effects on symptoms of cognitive dysfunction on any ECT occasion. This is in agreement with clinical experience that symptoms of cognitive dysfunction, when secondary to affective disorders, mainly respond early in an ECT series. Clinical outcome of this factor was linked to acute EEG slowing following ECT 3–4, similar to that of the anxiety-agitation factor (Table 3).

Concluding Remarks

The results of this investigation showed that in depression the different dimensions of emotion and cognition had different neurophysiological correlates and also a different time-course of recovery during ECT. The meaningfulness of the clinical differentiation in depressive symptom clusters (depression-retardation and self-depreciation) and symptoms of anxiety-agitation was supported by the correlations with neurophysiological changes and the differences in the clinical course following ECT. Acute EEG slowing and CBF decrease, although correlating with acute clinical improvement, could be interpreted as reflecting a post-ictal depression of brain function, presumably representing an unavoidable side-effect of ECT. These acute neurophysiological changes could not be related to clinical outcome. Thus, it is not the depressant acute effect of the epileptic seizure that reflects mechanisms important for the clinical recovery. The clinical evaluation following two or three treatments (pre-ECT 3–4) carried valid predictive information about the outcome (most evident regarding symptoms of depression-retardation, self-depreciation, and cognitive dysfunction), which is in close agreement with current clinical experience. Regarding the more gradual increase in EEG slowing a reverse relationship was found with clinical improvement, suggesting that this EEG effect during serial ECT represents another unwanted side-effect. Thus, it would seem likely that the gradual clinical as well as neurophysiological changes reflect entirely different effects upon the CNS than those found following single treatments.

The CBF level of depressed patients before treatment was somewhat higher compared to controls matched according to age and sex (Silfverskiöld et al. 1986). The relations found in this study between clinical improvement and CBF decrease (Tables 2, 3, and 4) need not indicate that serial ECT causes persistent brain dysfunction. On the contrary, symptom relief may rather reduce cortical arousal and the amount of "brain work", linked to emotional and intellectual disturbances, inherent in clinically depressed patients. Thus, the moderate non-acute CBF decrease may reflect a normalization of the patients' clinical condition.

The different components of the depressive syndrome have shown a relationship with global neurophysiological measurements, becoming especially evident by the way they react with the different time courses connected with serial ECT. The relation between the different clinical symptom clusters and the neurophysiological, neurochemical, and neurohumoral parameters must in the future also be analysed on a hemispheric and regional level.

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