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Effects of Electroconvulsive Therapy on EEG and Cerebral Blood Flow in Depression

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Summary. Changes in global EEG and global cerebral blood flow (CBF) and their relationship following electroconvulsive therapy (ECT) were studied in 21 depressed in-patients, examined before and after treatment during an ECT series and at follow-up. Two patterns of ECT action could be discerned: (1) acute changes related to single ECT's. The effects on CBF were more marked at the beginning of the ECT series, while the EEG slowing became more pronounced towards the end; (2) non-acute accumulating ECT effects which were insignificant for CBF, whereas the EEG slowing increased progressively during the ECT series. Thus acute and non-acute effects of ECT on EEG and CBF follow different patterns indicating independent seizure effects. They showed a different time course and few correlations were found, suggesting that CBF is more linked to cortical changes while EEG is probably more related to activity in deeper, subcortical structures.

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

Introduction

The induction of epileptic seizures for therapeutic purposes in mentally ill patients was first introduced by Meduna (1935) using camphor and later cardiazol. This technique has since been improved in different ways by using electrical stimulation (Cerletti and Bini 1938) and the introduction of muscle relaxant agents during brief narcosis. Today, 50 years later, electroconvulsive therapy (ECT) is still considered to be the superior treatment in severe depression. There is, however, still a great need for knowledge about the basic mechanisms of the positive and negative effects of this method. Lately, research has been intensified to study the course of illness and treatment by objective, neurophysiological methods such as EEG and regional cerebral blood flow (rCBF).

EEG, introduced by Berger (1929), has been widely used for monitoring immediate and more persistent effects of ECT (Meyer-Mickeleit 1949; Ottosson 1960; Weiner 1980). During the course of an ECT series, EEG shows a progressive decrease in the mean frequency along with an increase in the mean amplitude and the slowing becomes more persistent with increasing number of treatments (Chusid and Pacella 1952; Fink and Kahn 1957; Ottosson 1960). An increase of seizure duration amplifies the EEG effects (Meyer-Mickeleit 1949; Stein et al. 1968).

Bilateral (BL) ECT is usually considered to have a more marked effect on EEG than non-dominant unilaterally (UL) applied current (Chusid and Pacella 1952; Fink and Kahn 1957; Abrams et al. 1973; Marjerisson et al. 1975). However, Sand-Strömgren and Juul-Jensen (1975) found an equal amount of slowing with both stimulation techniques, after correction for shorter and incomplete seizures, which are more common in UL than BL ECT. Several authors have reported great inter-individual variability in amount, rate and persistence of EEG changes following ECT. Such changes have been related to the pre-treatment record, induction features, and to the number and frequency of seizures (Chusid and Pacella 1952; Green 1957, 1960; Stein et al. 1968; d'Elia 1970; Volavka et al. 1972; Robin and Tissera 1982). Following the termination of the ECT series, EEG slowing gradually disappears along with an increase in the mean frequency returning to a normal level, usually within 2–6 weeks (Klotz 1955; Weiner 1980). Only studies with quantification of EEG activity are relevant in this context.

Measurement of rCBF represents a more recent approach in the study of the functional state of the normal and diseased brain. Kety et al. (1948) found a moderate reduction of the cerebral metabolic rate and a marked (35%) CBF decrease following single ECT, while Wilson et al. (1952) reported a small insignificant flow reduction following an ECT series. Prohovnik et al. (1986) studied rCBF during ECT and found acute flow reductions where the flow patterns were related to the stimulation technique (low-dose UL versus BL ECT).

The aim of the present investigation was to study immediate and more long-lasting effects of ECT on EEG and CBF. Details of the post-ictal EEG changes have been published separately (Rosén and Silfverskiöld 1987). This article will describe the global changes and inter-correlations of EEG and CBF. The clinical symptomatology in depression and changes following ECT have been correlated with the global changes of EEG and CBF elsewhere (Silfverskiöld et al. 1987).

Patients and Methods

Patients

A total of 21 in-patients (7 men, 14 women) referred for ECT were studied by clinical ratings, rCBF, and EEG before and

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after single treatments and at follow-up. Non-dominant UL ECT is the standard mode of treatment at our clinic, but it was decided to give BL ECT during a limited period of time to enable a comparison to be made between the two techniques. The UL ECT group consisted of 13 patients (4 men and 9 women) with a mean age of 63.3 ± 11.6 years (range 44–83). In the BL ECT group there were 10 patients (4 men and 6 women) with a mean age of 64.7 ± 13.2 years (range 37–84). This group included 1 woman who received two treatment series with a 6-month interval. This patient and 1 man in the BL ECT group had been treated with UL ECT during an earlier depressive episode and are therefore present in both groups. The data are thus based on 13 series of UL and 11 series of BL ECT in 21 patients.

Two trained senior psychiatrists made the psychiatric diagnoses according to DSM-III (1980), first separately followed by a final diagnostic agreement. Most patients in both ECT groups belonged diagnostically to the group of major affective disorders (9 in the UL and 5 in the BL ECT group). Other diagnoses were bipolar disorder (1), atypical bipolar disorder (5), dysthymic disorder (1), atypical depression (1), and atypical psychosis (1). None of the patients had received ECT within 12 months prior to the investigation, except for 1 case who relapsed and was treated again with BL ECT after 6 months. All patients reported right-handedness. Patients with neurological deficits, major somatic disorder, chronic psychosis or addiction, were excluded from the study; 1 patient with bipolar disorder was on prophylactic lithium medication during the trial. During the ECT series, the patients did not receive tricyclic anti-depressants (TAD), barbiturates, benzodiazepines, or any other drug known to interfere with ECT or with the CBF pattern. One patient in the BL ECT group suffering from an atypical psychosis was given an anti-psychotic dose of neuroleptics (Haloperidol) at the beginning of the ECT series. At follow-up, two patients in each of the groups were taking a moderate to a low dose of TAD.

Methods

EEG. The EEG recordings were made in the clinical neurophysiological department situated nearby by a skilled EEG technician with due attention being given to eye movements, muscle and other physiological and technical artefacts. A routine EEG recording was made each time with a Siemens-Elema Electroencephalograph with the patient resting in a supine position, awake but with eyes closed. Effects on the EEG of eye opening, hyperventilation for 5 minutes, and intermittent light were recorded.

On each occasion, a quantitative frequency analysis was made of the EEG from four bipolar channels, symmetrically placed over pre-central and parietal areas (F3-C3; C3-P3; F4-C4; C4-P4). Computations were made, using a fast Fourier transform (FFT) algorithm (the BrainLab program, Digital on a PDP 11/03 computer). The time constant of EEG amplifiers was 0.3 s and the low pass filter set at 70 Hz. During the sampling period, individual subspectra from 30 s of sampling time were monitored on-line on a screen. This allowed individual spectra to be excluded from the final averaging if periods of artefacts occurred during the sampling. A final FFT spectrum was averaged from 20 individual subspectra, making the total sampling time per spectrum 10 min. From the FFT spectra the total EEG power (pW) within the frequency range of 0.5–25 Hz was automatically calculated as well as its distribution within the delta 0.5–4 Hz, theta 4.1–8 Hz, alpha 8.1–13 Hz and beta 13.1–25 Hz bands (%). From these figures the absolute EEG power (pW) within each frequency band was calculated. In order to simplify the data management, we calculated the EEG power within the 0.5–8 Hz range (low frequency power, LFP). The EEG variable LFP showed very large inter-individual variations (range of LFP: 9.36–224.2 pW). In order to simplify calculations, LFP at the first examination (before the first ECT) was taken as a reference and following values expressed as multiples of this (low frequency power index, LFPI). As this investigation was concerned with generalized EEG and CBF effects of ECT, the sum of EEG power from each of the four EEG channels was used in the analysis. The frequency of EEG activity within each frequency band having the highest power values was also automatically determined. The frequency of this dominant EEG activity (DF) was used as an independent variable. It was expressed as the average of the four EEG channels from which the recordings were taken. Typical effects of ECT on spectrum analysis EEG in a depressed patient are shown in Fig. 1. In this case, the acute effect was a leftward shift of DF (from 8.6 to 7.8 Hz) and the non-acute effect of ECT (from pre-ECT 1 to pre-ECT 5–7) appeared as an increase of LFPI (from 1.00 to 4.25).

rCBF. rCBF was measured during rest in 16 regions of each hemisphere by the ^{133}Xe inhalation technique (Obrist et al. 1975; Risberg et al. 1975). A detailed description of the measurement system (NDS-inhalation-cerebrograph) and the methods of curve analysis have been presented elsewhere (Risberg 1980). In the present study the initial slope index (ISI), (Risberg et al. 1975) was used. This index of mainly grey matter blood flow was selected because of its high reliability. ISI was expressed as the mean of both hemispheres corrected for pCO_2 to 40 mm Hg (0.75 ISI/mm Hg, Maximilian et al. 1980). Arterial pCO_2 (pCO_2) was estimated from end-tidal values of expired CO_2 concentrations.

ECT. Conventional ECT equipment (Siemens Convulsator 622) and procedure were used. The electrode placement was either bifronto-temporal (BL) or right non-dominant UL, as described by d'Elia (1970). Atropine, methohexital and suxamethone were used during brief narcosis. The amount of muscle relaxant was chosen so as to make the switch from the tonic to the clonic phase visible. This moment was selected as the starting point for measuring the seizure duration (by stopwatch) until no more signs of the seizure could be seen. Treatments were given 2–3 times weekly. The clinically observed duration of seizure, the amount of current used to elicit a seizure (stimulus energy in Joule), and the amount of methohexital and suxamethone given on each occasion were recorded.

Sham ECT. Four of the patients in the present material and ten patients from an extended patient group (Silfverskiöld et al. 1986) were investigated following "sham ECT", i.e. the full treatment procedure was performed but with no current passing between the electrodes. EEG and/or rCBF were recorded as in the main study.

Design. The design of the study was the basis for information to the patients. Approval was obtained from the Ethical Board of the University of Lund.

The patients were examined immediately before and 1–3 h after ECT on three different occasions during the ECT series:

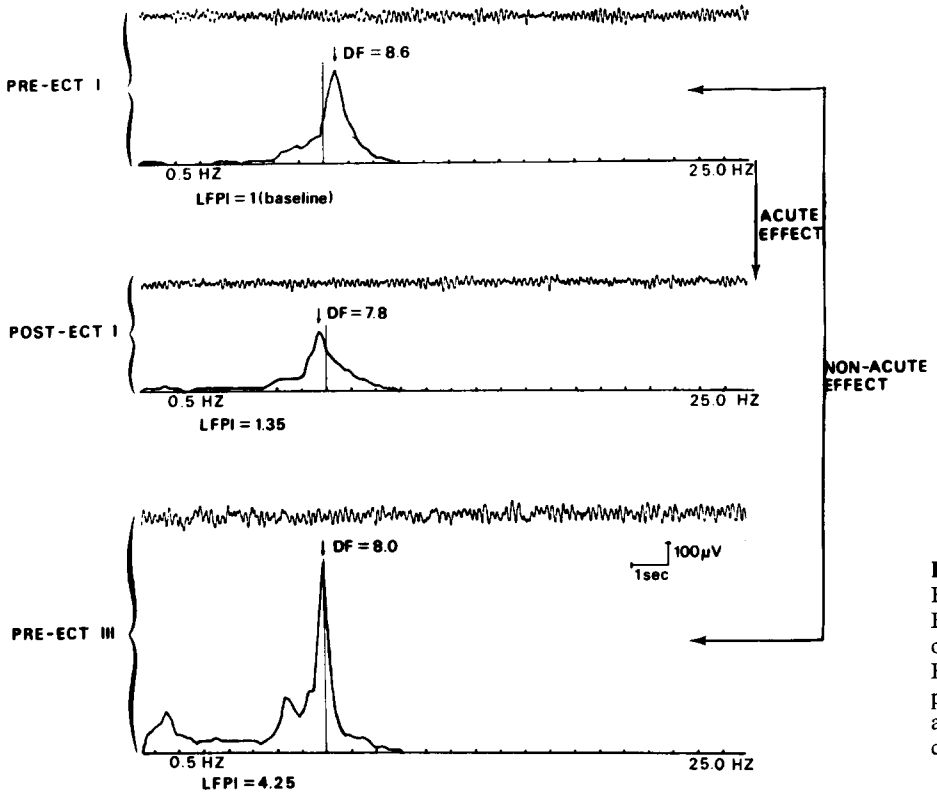

Fig. 1. ECT effects on spectrum analysis EEG in one depressed patient. Original EEG and FFT analysis records (C3-P3) from one patient pre- and post-ECT 1 and pre-ECT 5–7. Note acute leftward shift of the peak frequency (DF) following ECT 1 and increase of LFPI pre-ECT 5–7 as compared to pre- and post-ECT 1

ECT 1, ECT 3 or 4 (ECT 3–4), and ECT 5, 6 or 7 (ECT 5–7) and at follow-up 4–9 months after the ECT series.

When the patients were examined after single treatments, they were fully awake and oriented. The average delay between treatment and post-ECT CBF measurement was 108 ± 26 min. For EEG, this interval was 170 ± 40 min. An analysis of 72 measurements of EEG and CBF changes following ECT in relation to time lapse did not reveal any significant correlation (time lapse correlated with Δ CBF $r = 0.19$; time lapse correlated with Δ LFPI $r = 0.11$).

Statistics. The paired *t*-test was used to evaluate changes during the course of treatment. The Spearman rank correlation coefficient test was applied to evaluate correlations between changes in various variables during the ECT series. The one-way ANOVA test was used to evaluate differences between separate groups of subjects (i.e. UL vs BL ECT).

Table 1. EEG and cerebral blood flow (CBF)(mean global values and correlations) in depression before electroconvulsive therapy (ECT)

	Absolute values		Correlations	
		n	ISI	*n*
Log LFP (low frequency power)	1.72 ± 0.29	23	0.27	23
DF (dominant frequency)	9.19 ± 1.03	23	0.41	23
ISI (initial slope index)	50.25 ± 8.10	24		

Results

Results based on the analysis of four variables will be presented: LFPI, DF, ISI, and seizure duration. Results from the combined UL and BL groups will be described followed by an analysis of the differences between groups regarding the two treatment techniques. The numbers of patients show occasional variation due to incomplete data.

Before the first ECT all patients by and large had normal EEG and CBF findings with regard to age. DF and ISI showed a weak positive correlation (Table 1).

The effects of ECT will be discussed as acute and non-acute changes. The acute effects were differences between registrations before and after a single treatment (post- minus pre-ECT). Non-acute effects were differences between the pre-treatment situation before the first ECT and pre-ECT 3–4 and pre-ECT 5–7 (pre-ECT 3–4 minus pre-ECT 1; pre-ECT 5–7 minus pre-ECT 1).

Acute Effects of Single ECT's

Changes in EEG and CBF following ECT are presented in Fig. 2.

LFPI showed only a minor increase at ECT 1. The response then gradually increased with number of treatments. The reduction in DF followed a similar pattern during the series. The pattern of ISI changes was clearly different from that of the EEG variables and the effects were different both early and late in the series. ISI showed a marked decrease at ECT 1, followed by gradually less pronounced changes at ECT 3–4 and 5–7.

The mean seizure duration, stimulus energy (Joule), and the amount of barbiturate (methohexital) were not found to vary systematically during the ECT series. The seizure dura-

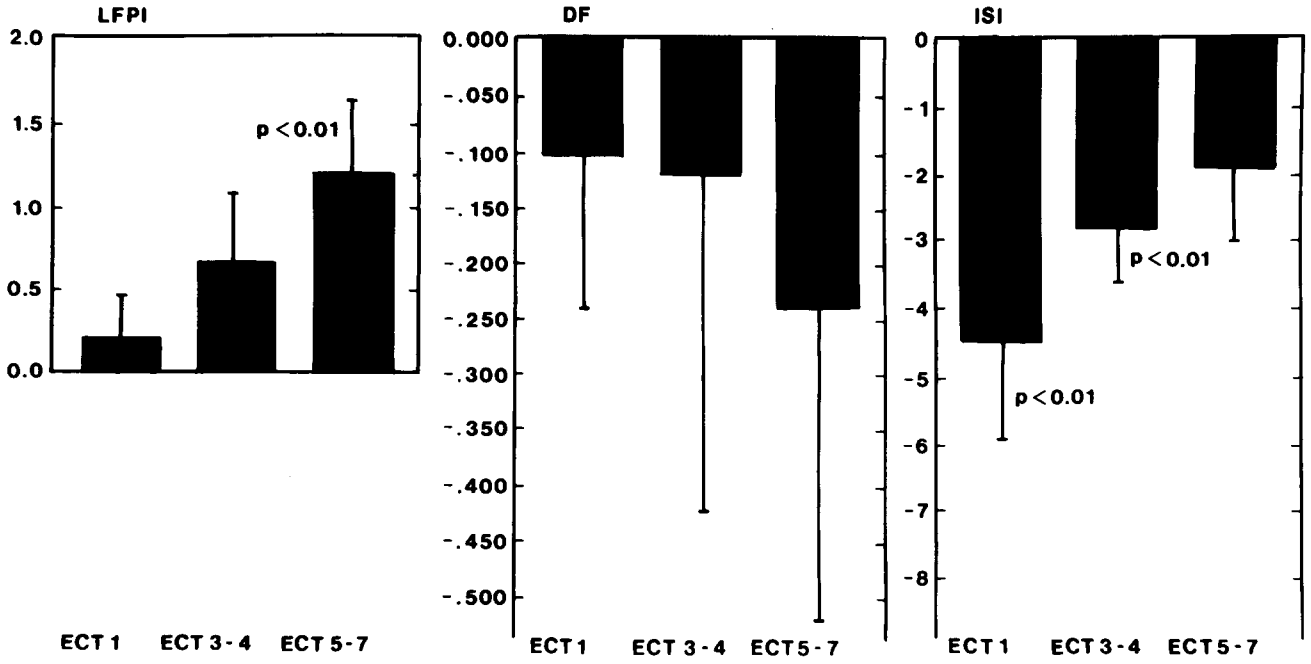

Fig. 2. Acute ECT effects on EEG and CBF in depression. Means and standard errors of the mean are given as well as *P* values (paired *t*-test). ECT 1 $n = 20$; ECT 3–4 $n = 18$; ECT 5–7 $n = 19$

Table 2. Acute effects of ECT in depression. Correlations between changes (post/pre-ECT) in EEG (LFPI, DF), CBF (ISI) and seizure duration

	ECT 1 ($n = 20$)		ECT 3–4 ($n = 16$)		ECT 5–7 ($n = 18$)	
	ISI	Seizure duration	ISI	Seizure duration	ISI	Seizure duration
LFPI	0.36	0.15	−0.55*	0.65**	−0.15	0.47*
DF	0.02	−0.24	0.07	−0.42	0.39	−0.31
ISI		−0.25		−0.38		0.03

* $P = 0.05$ (two-tailed)
** $P = 0.01$ (two-tailed)

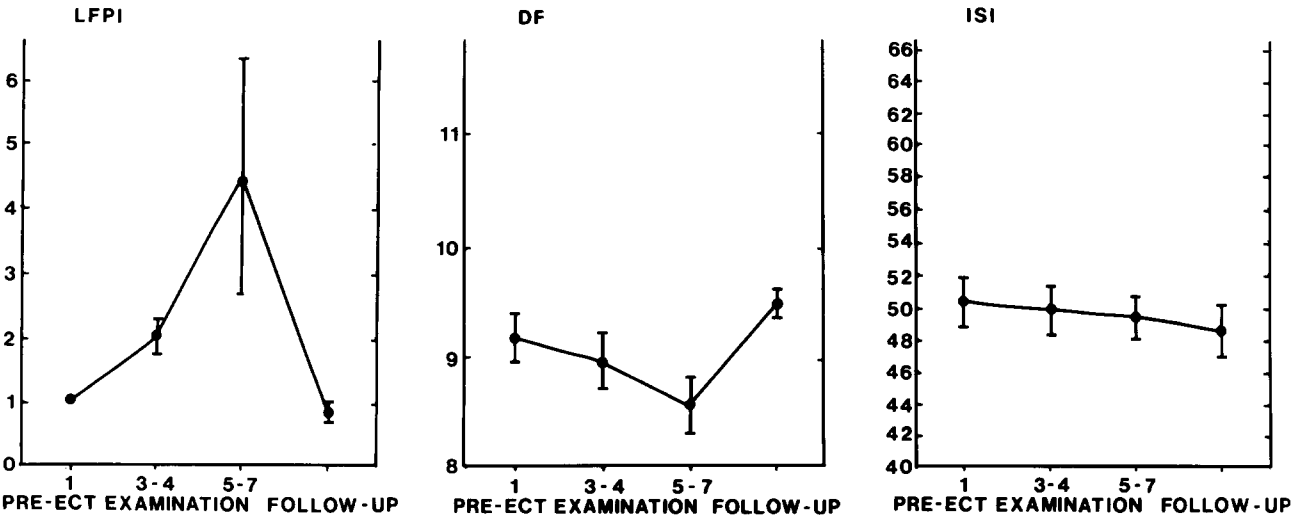

Fig. 3. Non-acute ECT effects on EEG and CBF during serial ECT in depression and at follow-up. Means and standard errors of the means are given as well as *P* values (paired *t*-test). ECT 1, ECT 3–4, ECT 5–7 $n = 23$; follow-up $n = 19$

Table 3. Non-acute effects of serial ECT in depression. Changes in EEG (LFPI, DF) and CBF (ISI)

	Pre-ECT 3–4 – pre-ECT1		Pre-ECT 5–7 – pre ECT1	
		n		*n*
LFPI	0.96 ± 1.42**	23	3.50 ± 9.18*	23
DF	−0.45 ± 0.72**	22	−0.63 ± 0.87**	23
ISI	−0.43 ± 6.82	21	−1.50 ± 6.57	23

* $P<0.05$
** $P<0.01$

Table 4. Non-acute effects of ECT in depression. Correlations between changes in EEG (LFPI, DF), CBF (ISI) and accumulated seizure duration

	ISI		Accumulated seizure duration	
	Pre-ECT 3–4 –pre-ECT1 ($n=20$)	Pre-ECT 5–7 –pre-ECT1 ($n=23$)	Pre-ECT 3–4 –pre-ECT1 ($n\pm20$)	Pre-ECT 5–7 –pre-ECT1 ($n=23$)
LFPI	0.06	0.02	0.20	0.62**
DF	0.22	0.19	0.07	−0.34
ISI			0.07	0.10

** $P<0.01$ (two-tailed)

tion (in s) at ECT 1 was 51 ± 13, at ECT 3–4 47 ± 16, and at ECT 5–7 49 ± 13. The stimulus energy (Joule) at ECT 1 was 31.8 ± 9.5, at ECT 3–4: 40.4 ± 23.3, and at ECT 5–7 38.0 ± 17.8. The amount of methohexital (mg) given at ECT 1 was 66.8 ± 26.1, at ECT 3–4 62.8 ± 15.7, and at ECT 5–7 61.8 ± 14.8.

Stimulus energy and barbiturate dose showed no significance as background factors in relation to EEG and CBF variables.

The correlations between changes in LFPI, DF, ISI from before to after ECT and seizure duration are shown in Table 2. The correlations between LFPI and ISI varied during the ECT series, turning negative and significant at ECT 3–4. This means that an increase in slow waves was accompanied by a decrease in CBF. The correlations between changes in LFPI and seizure duration were positive during the ECT series, reaching a significant level at ECT 3–4 and ECT 5–7. Thus, the longer the duration of the seizures, the more marked was the increase in slow waves. No other significant relationships between the variables were found.

Non-Acute Effects During the ECT Series

Changes in EEG and CBF are shown in Fig. 3 and Table 3. LFPI showed a significant increase from pre-ECT 1 to pre-ECT 3–4 and increased even further to pre-ECT 5–7. At the follow-up examination, the LFPI was somewhat below the pre-treatment level. DF showed a progressive decrease from pre-ECT 1 to pre-ECT 3–4 and pre-ECT 5–7. At follow-up, the DF value was slightly higher than the pre-ECT 1 level. The ISI level showed a minor, insignificant progressive decrease during the ECT series and at follow-up. The mean of the accumulated seizure duration from the first until pre-ECT 3–4 was 135 ± 35 s and from the first until pre-ECT 5–7 was 213 ± 57 s.

Correlations between non-acute changes of LFPI, DF, ISI, and seizure duration are shown in Table 4. The significant correlation between LFPI and seizure duration at pre-ECT 5–7 indicated that an increase in the accumulated seizure duration was accompanied by an increase in the EEG slowing.

Comparison Between UL and BL ECT

Before treatment, no significant difference between the UL and the BL groups was found regarding EEG. The BL group showed, however, a slightly higher (NS) ISI level (ISI 52.88) compared to the UL group (ISI 48.01). After the first treatment, this difference decreased to about 2 ISI units and remained at this level during the rest of the series including follow-up. The flow decrease from before treatment to follow-up was more marked in the BL ($P<0.06$) than in the UL ECT group.

The EEG variables did not show any significant differences between the treatment techniques at any stage. At follow-up, however, the UL group showed significantly less LFPI ($P<0.01$) compared to its pre-treatment value. No such difference was found for the BL group.

The acute effects of ECT on the two groups were also compared. The decrease in ISI following the first treatment was greater in the BL group (−8.04) than in the UL group (−0.77). The difference between the groups was significant ($P<0.01$). Such a difference was not found later in the ECT series. No other significant differences regarding the physiological variables were found between the groups.

Sham ECT

The results of the four patients examined by EEG and rCBF did not reveal any significant differences from before to after "treatment" (Δ LFPI −0.20 ± 0.17; Δ DF −0.04 ± 0.15; Δ ISI 1.73 ± 6.54). Neither did we find any significant CBF change from before to after simulated ECT in the ten patients from the extended patient group (ISI before 54.19 ± 7.64 and after 57.62 ± 7.60).

Discussion

The results of this study show clear effects of ECT on global EEG and CBF. We have tried to evaluate whether the changes recorded could to any extent be explained by other components of the ECT procedure than the induced seizure, e.g. narcosis. Only minor EEG changes and a tendency towards an increase in CBF were noted following sham ECT, contrary to the results in the main series. Our conclusion that the narcosis per se cannot explain the present findings, is further supported by the lack of significant correlations between the neurophysiological measures and the amount of barbiturate administered.

The more marked CBF decrease following the first ECT in the BL group, compared to the UL group, has to be interpreted cautiously; no corresponding effects were seen in the EEG. To some extent, this finding could be related to the higher pre-treatment flow level in the BL group, but it may also indicate a stronger cortical effect induced by the BL than by the UL technique at the beginning of a series.

The present analysis focused on a comparison between EEG and CBF effects of ECT. It seems important to differentiate between immediate effects of the epileptic seizure and changes attributed to accumulating post-ictal effects. The results also indicated that the acute effects of ECT on EEG and CBF follow different patterns. The slowing of EEG, as reflected by the increase in LFPI and the decrease in DF, was small following the first ECT and became more marked later in the series. The first ECT caused a marked decrease in CBF, whereas the CBF decreases later became less marked. The differences between the EEG and CBF effects cannot be explained by any systematic differences in the time lag between treatment and EEG or CBF measurements. Presumably, the changes reflect general changes in the immediate response patterns of the brain during the course of ECT.

In most clinical and experimental studies, a correlation has been found between a decrease in CBF, usually an indication of a decrease in the cerebral metabolism, and a decrease in the mean frequency of EEG (Sulg 1969; Ingvar et al. 1976, 1979). There are, however, exceptions with an uncoupling of the EEG/rCBF relation as in luxury perfusion states after cerebrovascular lesions (Lassen 1966) and following induced hypoxia (Freeman and Ingvar 1967). It is a well established fact that post-ictally there is a decrease of CBF and metabolism (Kety et al. 1948; Bolwig et al. 1977; Silfverskiöld et al. 1979, 1984, 1986; Engel 1983) and a decrease in the mean frequency of EEG irrespective of what mechanism elicited the generalized seizure activity. The results of our study are in agreement with the general relationship between EEG slowing and decrease in CBF. However, the differences in the time course of the changes indicate that the measurements reflect independent, post-ictal pathophysiological events. This is further supported by the inconsistent correlations between EEG and CBF during the course of ECT, being significant only as an acute effect between LFPI and ISI at ECT 3–4 (Table 4). This lack of correlation earlier and later during the series may be related to the different acute ECT effects on LFPI and ISI at these stages. If any correlation exists, it should be expected when the changes are most pronounced in both variables simultaneously, thus at ECT 3–4.

An important question is whether autoregulation of CBF and coupling between CBF and metabolism are retained at the time of the post-ictal CBF measurement. During a generalized seizure, the cerebral metabolism and CBF are markedly (110%–200%) increased (Plum et al. 1968; Brennan and Plum 1970; Ingvar 1982) followed post-ictally by a period of decreased metabolism and returning to the pre-seizure level within 15 min (Brodersen et al. 1973). Autoregulation of CBF returns within 60 min (Brennan and Plum 1970). Thus, the post-ECT CBF measurements in this study were performed after the restoration of cerebral metabolism and autoregulation. This assumption is supported by the absence of a significant correlation between the time lapse between ECT and CBF measurements and the changes of CBF.

Concerning the non-acute effects, marked changes in EEG occurred with no significant changes in CBF. This represents an unusual situation of marked slowing of EEG not accompanied by a decrease in CBF. Assuming that CBF reflects the level of cortical neuronal function and energy metabolism, the results indicate that the cerebral cortex does not undergo persistent changes following ECT. Available neurophysiological data indicate that the thalamus is the dominating pace-maker of rhythmic cortical activity as reflected in the EEG (Andersen and Andersson 1968; Niedermeyer and Lopez da Silva 1982). The thalamo-cortical projections are influenced by reticular systems, especially via the non-specific ventro-medial thalamic nuclei and the nucleus reticularis thalami (Schlag 1974; Scheibel 1980; Steriade et al. 1980). As demonstrated in this study, marked EEG effects, not accompanied by CBF changes, seem to give further support to the hypothesis that the specific therapeutic EEG effects are not primarily linked to cortical events but rather to deeper structures, possibly the reticulo-thalamic systems.

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