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Quantification of EEG Changes Following Electroconvulsive Therapy in Depression

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Abstract. Frequency analysis of EEG was made during electroconvulsive therapy (ECT) in patients with depression. Acute effects were quantified by calculating differences of EEG power from before to after the induced seizure, and were found to correlate with the duration of the seizure but not with the time lapse following the seizure. Increases in delta power were much more pronounced at the end of the treatment series than at the beginning. Non-acute effects were quantified as the differences from before the first treatment to the pre-ECT EEG later in the series. Increases in delta power correlated with the accumulated seizure duration and positively with the time lapse from the previous seizure, suggesting that it takes considerable time for this effect to develop. The concept of two different sources of EEG slowing during the ECT series is supported by different correlations between acute and non-acute EEG slowing on the one hand and on the other symptoms of depression, anxiety, cognitive disturbances, and cerebral blood flow as presented elsewhere.

Key words: ECT – EEG – Depression

Introduction

It is a well-known phenomenon that a generalized epileptic seizure produces an immediate post-ictal depression and slowing of the EEG (Gibbs and Gibbs 1952). Factors influencing the degree and duration of the post-ictal EEG slowing are, however, not known, partly due to the fact that the duration and exact time of clinical seizures are usually not known in detail. Such information can be obtained from intentionally induced seizures such as electroconvulsive therapy (ECT). EEG has been monitored extensively in ECT studies (Fink 1979). During the course of an ECT series, EEG shows a progressive slowing and an increase in amplitude (Chusid and Pacella 1952; Fink and Kahn 1957; Ottosson 1960). Following termination of the ECT series, EEG gradually returns to the habitual pattern, usually within a few weeks (Klotz 1955; Weiner 1980).

Methods have been developed over recent years allowing an exact quantification of the EEG activity (Lopez da Silva 1982). The aim of the present study was to describe acute and more long-lasting effects on quantified EEG of electrically induced generalized seizures, which can be exactly defined in time and duration.

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Patients

The data were based on 13 series of right non-dominant unilateral (UL) and 11 series of bilateral (BL) ECT in 21 patients. Diagnostically the patients belonged to the group of major affective disorders in accordance with the DSM-III classification (Silfverskiöld et al. 1987a and b). In the UL ECT group there were 13 patients (4 men and 9 women) with a mean age of 63 ± 12 years (range 44–83). In the BL ECT group there were 10 patients (4 men and 6 women) with a mean age of 65 ± 13 years (range 37–84). The BL ECT group included 1 woman, who had received 2 series of ECT with an interval of 6 months. 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 patients had not 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. Patients with major somatic disorders, neurological deficits, chronic psychosis, or addiction were excluded from the study. Tricyclic anti-depressants, barbiturates, or benzodiazepines were not used during the series. The study was approved by the Ethical Board of the University of Lund.

Methods

The EEG recordings were made by a skilled EEG technician with due attention to eye movements, muscular and other physiological and technical artefacts with a 16-channel Siemens-Elema Electroencephalograph (time constant 0.3 s, low pass filter: 70 Hz). Quantitative frequency analysis of EEG from four bipolar channels symmetrically placed over pre-central and parietal areas (F3-C3; C3-P3; F4-C4; C4-P4) was made on each occasion using a Fast Fourier Transform (FFT) algorithm (The Brain-Lab program, Digital on a PDP 11/03 computer). During the sampling period, individual subspectra from 30-s samples were monitored on-line on a screen allowing individual spectra with artefacts to be excluded from the final averaging. A final FFT spectrum was averaged from 20 individual subspectra making the total sampling time per spectrum 10 min. The total EEG power (pW) within the frequency range of 0.5–25 Hz was automatically calculated as well as the proportions of power within 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.

Table 1. Seizure duration and ECT: post-ictal EEG interval

	ECT 1		ECT 3–4		ECT 5–7	
	Mean	Range	Mean	Range	Mean	Range
Seizure duration (s)	51	26– 75	47	28– 99	47	25– 94
Interval between ECT and post-ictal EEG (min)	178	123–277	169	85–389	162	126–198

Table 2. Accumulated seizure duration and previous seizure interval

	ECT 3–4		ECT 5–7	
	Mean	Range	Mean	Range
Accumulated seizure duration before the pre-ECT EEG (s)	135	82–218	215	138–386
Interval between pre-ECT EEG and previous seizure (h)	46	33– 71	40	28– 69

Design. Following informed consent the patients were examined immediately before and 1 to 3 h after ECT on three different occasions during the ECT series: ECT 1, ECT 3 or 4 (ECT 3–4), and ECT 5, 6 or 7 (ECT 5–7) and at follow-up 4 to 9 months after the ECT series. When the patients were re-examined following single treatments they were fully awake and oriented. The duration of the seizure was recorded by the patient's clinical signs. Atropine, methohexital, and a muscle relaxant 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 the starting point for measuring the seizure duration (by stop-watch) until no more signs of the seizure could be seen. The intervals between ECT and the post-ictal EEG recording on each occasion and the seizure durations are shown in Table 1. The intervals between the pre-ECT EEG recording and the previous seizure in the series and the accumulated seizure duration during the series before the actual pre-ECT EEG recording are given in Table 2. The seizure effects on the EEG were studied from two aspects: as acute and non-acute changes. The acute effects were calculated as individual differences from before to after single treatments (post-minus pre-ECT) and the non-

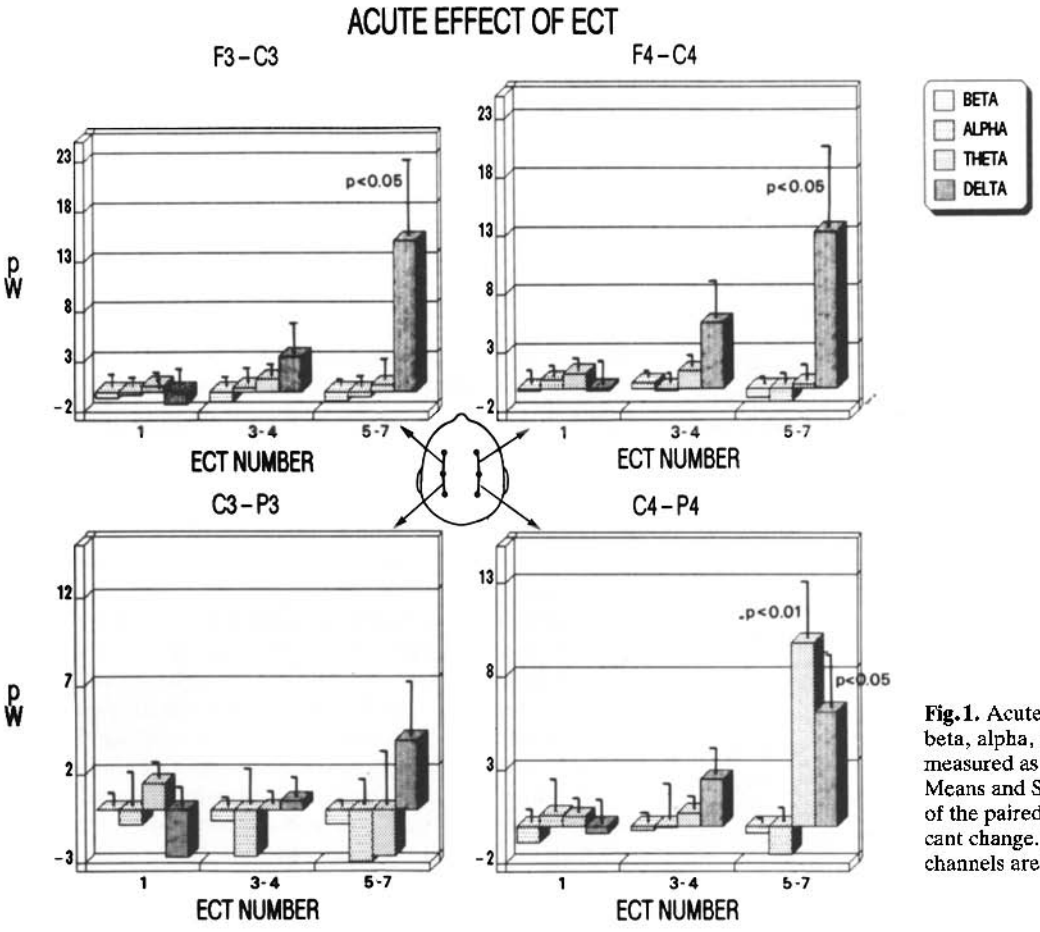

Fig. 1. Acute changes of EEG power in the beta, alpha, theta, and delta bands at ECT, measured as post-pre ECT differences. Means and SEM are given as well as results of the paired *t*-test, when indicating a significant change. The positions of the recording channels are indicated by the figurine

acute effects were measured from before the first ECT to pre-ECT record 3–4 and to pre-ECT 5–7 (pre-ECT 3–4 minus pre-ECT 1; pre-ECT 5–7 minus pre-ECT 1).

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 seizure durations and time intervals and ECT effects on EEG (measured as changes in delta power). Number of examinations less than 24 were due to missing values. The one-way ANOVA test was used to evaluate differences between groups receiving UL versus BL ECT.

Results

Acute effects of single ECTs

The calculated changes of absolute power within the four classical frequency bands are plotted in Fig. 1 for the four recording channels. Although the variability was high, the acute effects of the seizures changed progressively during the ECT series. After the first seizure (ECT 1) no appreciable EEG effects were seen. At the end of the series (ECT 5–7) the same seizure duration produced a significant increase in delta power, being more marked in the pre-central regions. In the middle of the series (ECT 3–4) intermediate post-ictal effects were found. The acute effects on alpha and beta (8.1–25 Hz) components were small.

The relations between the acute effects on delta power and seizure duration and time lapse between seizure and post-ECT EEG recording were analysed with Spearman rank correlations (Table 3). No consistant correlations were found at ECT 1. At ECT 3–4 and ECT 5–7 significant correlations appeared between delta power increase and seizure duration for three of the leads. However, contrary to what could be expected, no significant correlations appeared between the amount of delta power increase and the time lapse between seizure and post-treatment EEG (range 85–389 min) (Table 1).

The one-way ANOVA test of variance did not demonstrate any significant differences between the patient groups receiving UL and BL ECT.

Non-acute effects of ECT on EEG

The development of the EEG power spectrum during the course of ECT, apart from immediate post-ictal contributions,

Table 3. Acute effect of ECT: Spearman rank correlations

		Acute change in delta power				
		F3–C3	C3–P3	F4–C4	C4–P4	
ECT 1	Seizure duration	–0.13	0.23	–0.12	–0.22	($n = 23$)
	Interval ECT – post-ictal EEG	0.45*	0.20	0.13	0.20	
ECT 3–4	Seizure duration	0.31	0.65**	0.55*	0.72**	($n = 21$)
	Interval ECT – post-ictal EEG	0.12	0.13	–0.04	–0.02	
ECT 5–7	Seizure duration	0.41*	0.21	0.41*	0.41*	($n = 23$)
	Interval ECT – post-ictal EEG	–0.38(*)	–0.08	–0.33	–0.16	

** $P < 0.01$ two tailed
* $P < 0.05$ two-tailed
(*) $P < 0.05$ one-tailed

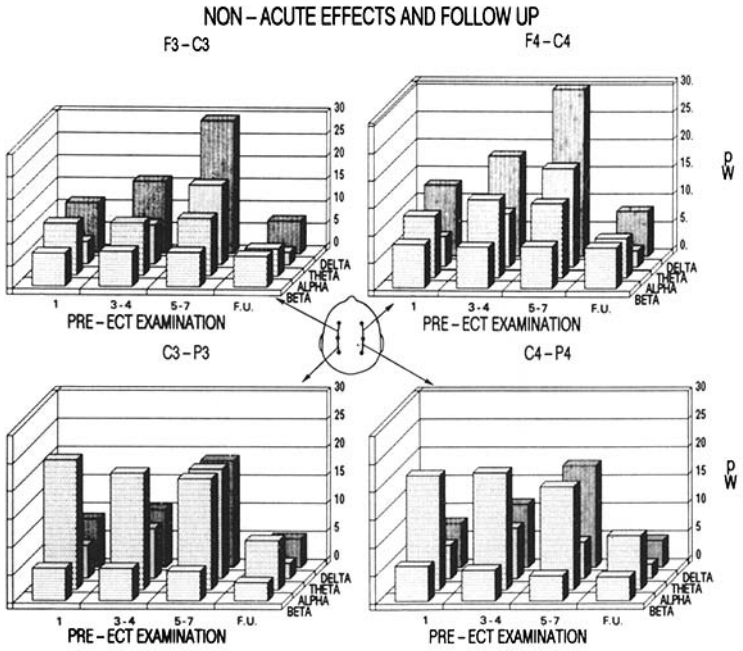

Fig. 2. EEG power (pW) for the four frequency bands at the three pre-ECT examinations and at the follow-up examination. Mean values are given. Non-acute differences are presented in more detail in Table 4. The positions of the recording channels are indicated by the figurine

Table 4. Non-acute differences in EEG power

			Delta (pW)	Theta (pW)	Alpha (pW)	Beta (pW)
Pre-ECT (3–4) minus pre-ECT	($n = 22$)	F3–C3	5.4 ± 2.5*	4.1 ± 1.0***	0.86 ± 2.3	0.6 ± 1.5
		C3–P3	1.8 ± 2.4	3.2 ± 0.9**	−2.1 ± 6.5	−0.1 ± 1.3
		F4–C4	5.8 ± 2.2**	4.5 ± 0.8***	3.6 ± 2.1	−0.2 ± 1.0
		C4–P4	3.5 ± 2.0*	3.2 ± 1.2**	0.9 ± 2.8	−0.8 ± 1.2
Pre-ECT (5–7 minus pre-ECT	($n = 22$)	F3–C3	20.2 ± 5.5***	13.6 ± 3.0***	2.7 ± 1.4*	0.8 ± 1.4
		C3–P3	11.1 ± 3.9**	14.4 ± 4.2**	−1.5 ± 7.2	−0.4 ± 1.1
		F4–C4	19.5 ± 5.3***	13.3 ± 3.0***	3.9 ± 1.3**	0.1 ± 1.5
		C4–P4	10.9 ± 3.3**	0.7 ± 1.8	0.1 ± 4.0	−1.6 ± 1.2
Follow-up minus pre-ECT	($n = 22$)	F3–C3	−4.0 ± 1.76*	−2.1 ± 0.8**	−5.0 ± 2.7*	−0.05 ± 1.7
		C3–P3	−3.7 ± 2.27	−3.1 ± 1.3*	−14.5 ± 10.4	−2.3 ± 1.2*
		F4–C4	−4.9 ± 2.13*	−2.2 ± 0.9*	−3.3 ± 1.5*	−0.13 ± 1.2
		C4–P4	−3.2 ± 2.15	−3.2 ± 1.3*	−10.0 ± 7.9	−1.7 ± 1.3

* $P < 0.05$; mean ± SEM
** $P < 0.01$
*** $P < 0.001$

Table 5. Non-acute effect of ECT: Spearman rank correlations

		Non-acute change in delta power				
		F3–C3	C3–P3	F4–C4	C4–P4	
Pre-ECT (3–4) minus pre-ECT	Accumulated seizure duration	0.20	0.28	0.15	0.17	($n = 21$)
	Interval since previous seizure	0.41(*)	0.35	0.18	0.16	
Pre-ECT (5–7) minus pre-ECT	Accumulated seizure duration	0.37(*)	0.43*	0.48*	0.46*	($n = 22$)
	Interval since previous seizure	0.48*	0.49*	0.45*	0.41(*)	

is illustrated in Fig. 2 by plotting the pre-ECT and follow-up values. The delta and theta power showed a gradual increase, more pronounced pre-centrally than post-centrally, and at follow-up returned to a level slightly below that found before the first treatment. The alpha and beta power did not show any major changes during the ECT series. At follow-up the post-central alpha power was lower than before the onset of treatment.

The non-acute effects of repeated seizures on the EEG were further evaluated by calculating individual differences between the pre-ECT examinations (3–4, 5–7) and the pre-ECT 1 values. These results are presented in Table 4 together with the differences between the follow-up examinations and the initial pre-ECT record. The non-acute increase of delta and theta power was significant at the pre-ECT 3–4 examination and became even more marked and significant at the pre-ECT 5–7 examination. At pre-ECT 5–7 there was also a significant increase in alpha power pre- but not post-centrally as compared to ECT 1.

At the follow-up examination the decrease in delta and theta power and also the decrease in alpha power pre-centrally were significant for most channels as compared with pre-ECT 1.

The relations between the non-acute changes in delta power in the four EEG channels and the accumulated seizure duration during the series until the time of examination, and the time lapse since the previous seizure (see Table 2), were evaluated using the Spearman rank correlation test (Table 5). No significant correlations appeared for changes at pre-ECT 3–4. At pre-ECT 5–7 there were significant and expected correlations between the accumulated seizure duration and delta power increase. Unexpectedly, there were consistent positive correlations between the time lapse from the previous seizure and the delta power increase, suggesting that it takes considerable time (within the range of 28 to 69 h, Table 2) for the non-acute delta power increase to develop.

The one-way ANOVA test did not reveal any significant differences between the UL and BL treated patients in terms of non-acute EEG effects or differences from pre-ECT 1 to follow-up.

Discussion

In the present study no significant differences in ECT effects on quantified EEG were observed between the UL and BL treated depressed patients. This justifies a common description of the whole group of 24 ECT series. Less EEG effects after UL stimulation than after BL stimulation have been reported (Chusid and Pacella 1952; Fink and Kahn 1957; Marjerrison et al. 1975) but have been questioned by others (Sand-Strömgren and Juul-Jensen 1975). Using quantitative assessment of the EEG, Kriss et al. (1978) showed preponderance of a slowing over the stimulated hemisphere for the initial 30 min after the seizure. No such asymmetries could be observed in our study which may be due to the longer delay between seizure and EEG recording. UL stimulation may also in our hands have elicited more generalized seizure activity compared to other investigators (Prohovnik et al. 1986).

The quantification of post-ictal EEG changes following electrically induced seizures may serve two purposes. Firstly, to establish the necessary prerequisites for making detailed correlations between quantified EEG, cerebral blood perfusion, and psychiatric symptoms during ECT (Silfverskiöld et al. 1987a and b), and secondly as a model for studying the post-ictal EEG more systematically than is usually possible following spontaneous epileptic seizures.

Most changes of the EEG frequency spectrum occurred in the low frequency range (delta and theta bands). The changes of delta and theta power occurred in parallel. Although the changes were more marked pre-centrally than post-centrally the patterns of changes were similar in all four recording channels, i.e. in the pre-central and post-central regions bilaterally. In order to simplify the analysis of the global post-ictal EEG effects following ECT and their correlations with cerebral blood flow and clinical changes, the overall power within the 0.5–8 Hz range was used (Silfverskiöld et al. 1987a and b).

Our results showed that acute effects (within 2–5 h) of generalized seizures of 0.5–1 min duration were dependent upon whether the seizure occurred in isolation, when almost no changes could be detected, or if it occurred as one in a series, when marked effects were found.

This gradual change of the brain's responsiveness to seizures was also reflected by the fact that the EEG did not return to its initial frequency content, even if as long a time as 70 h was allowed to pass since the previous seizures. This gradual change of the EEG measured as increase in delta power, was at least as large as the acute increase after a solitary seizure. The mechanisms behind this non-acute change of EEG power are probably different from the immediate post-ictal effects. This is supported by the observation that although the EEG slowing was dependent on the accumulated seizure duration preceding the recording, it became more marked the more time that had passed since the previous seizure. To date one can only speculate that this type of EEG slowing may be related to gradual changes of central neurotransmitter functions (Barkai 1986) or possibly kindling phenomena (Sackeim et al 1986).

The reason for the absence of an expected negative correlation between acute post-ictal slowing and time lapse between seizure and EEG recording is probably that the post-ictal observation periods (85–389 min) were too long to catch the immediate post-ictal EEG effects of the seizure which to a large extent fades away within 30 min (Kriss et al. 1978). At the time of our post-ictal recording the fading post-ictal effects were probably superimposed on a growing accumulating EEG effect of the repeated treatments. The concept of two different sources of EEG slowing during the ECT series is supported by the different correlations between acute and non-acute EEG slowing and clinical variables such as symptoms of depression, psychomotor retardation, self-depreciation, anxiety-agitation, and cognitive dysfunction described elsewhere (Silfverskiöld et al. 1987a and b).

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