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Self-Regulation of Slow Cortical Potentials in Psychiatric Patients: Alcohol Dependency[1]

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Ten unmedicated alcohol-dependent male inpatients participated in a Slow Cortical Potential (SCP) self-regulation task utilizing biofeedback and instrumental conditioning. These patients were hospitalized for treatment of alcohol dependency after chronic abuse of alcoholic beverages. Somatic withdrawal symptomatology had occurred recently and the patients were free of any withdrawal symptoms of the autonomic nervous system. Immediately after hospitalization patients were unable to control their SCPs without the reinforcement of immediate feedback across 4 sessions. Seven patients participated in a fifth session an average of 4 months later. Six out of these 7 patients had not had a relapse at the follow-up. In the fifth session these patients were immediately able to differentiate between the required negativity and negativity suppression, whereas the seventh patient, who had relapsed, was unable to control his brain potentials successfully. Results are further evidence that some of the frontocortical dysfunctions in alcohol-dependent patients are reversible. This could covary with a morphological restitution of the cortex.

Descriptor Key Words: slow cortical potentials; alcohol dependency; biofeedback; instrumental learning; CNV.

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As reported in Parts I and II of this publication series (Schneider, Rockstroh, Heimann, Lutzenberger, Mattes, Elbert, Birbaumer, & Bartels, 1992; Schneider, Heimann, Mattes, Lutzenberger, & Birbaumer, 1992) we consider slow-varying shifts of surface recorded EEG (SCPs) to represent neuronal excitability in the underlying cortical networks. The area specific regulation of excitability is thought to indicate the direction of preparatory attention (Elbert, in press; McCallum, 1988; Rockstroh, Elbert, Canavan, Lutzenberger, & Birbaumer, 1989). Therefore the investigation of the self-regulation of SCPs may serve to establish some mechanisms required for attentive behavior. The findings reported in Part I demonstrated such impaired self-regulatory mechanisms in patients with schizophrenia (Schneider, Rockstroh et al., 1992). In this study subjects were required to modify their SCPs upon command. In a biofeedback design, position and movement of a visual feedback stimulus represented the change in SCPs during the 8-s feedback interval. Schizophrenic patients, unlike control subjects, showed no significant differentiation between volitional negativity increase and negativity suppression in the first few sessions, but achieved SCP-control after extensive training (sessions 18-20). In contrast to the schizophrenic patients, depressives quickly learned to regulate their SCPs and to maintain this ability across 20 sessions (Schneider, Heimann et al., 1992).

Several studies have demonstrated similar endogenous event-related potentials as the reduced CNV for schizophrenics, alcoholics, and alcohol-intoxicated healthy controls (Kopell, Tinklenberg, & Hollister, 1972; Rohrbaugh et al., 1988; Skerchock & Cohen, 1984; Timsit-Berthier, Gerono, Rousseau, Mantanus, Abraham, Verhey, Lamers, & Edmonds, 1984). The reduced CNV may suggest reduced attentional preparation, which in connection with perseveration tendencies observed in alcoholics may point to a frontocortical deficit, as has been shown by neuropsychological and morphological studies (Berglund et al., 1987; Corbett & Harper, 1988; Fitzhugh, Fitzhugh, & Reitan, 1965; Götze, Kühne, Hansen, & Knipp, 1978; Harper, Kril, & Karpy, 1987; Harper, Kril, & Daly, 1988; Jones & Parsons, 1971; Oscar-Berman & Ellis, 1987; Sachs, Russel, Christman, & Cook, 1987; Samson, Baron, Feline, Baries, & Crouzel, 1986; Tarter, 1975). Investigations of alcohol-dependent patients thus may contribute to our understanding of frontal lobe functioning.

Follow-up studies of alcohol-dependent patients with a longer period of abstinence are especially important, since several studies have shown morphological, neurophysiological, and neuropsychological restitution of the cortex even after extensive periods of abuse (Artmann, Gall, Hacker, & Herrlich, 1981; Cala et al., 1983; Carlen, Wortzman, Holgate, Wilkinson, & Rankins, 1978; Grünberger & Maly, 1972; Mann, Optiz, Petersen,

Schroth, & Heimann, 1989; Muuronen, Bergman, Hindmarsh, & Telakivi, 1989; Porjesz & Begleiter, 1985; Ron, Acker, Shaw, & Lishman, 1982; Schroth, Naegele, Klose, Mann, & Petersen, 1988; Templer, Ruff, & Simpson, 1975).

The aim of the present study was to show that chronic alcohol-dependent patients, like other patients with frontal lobe disorders (Birbaumer, Elbert, Canavan, & Rockstroh, 1990), demonstrate a reduced ability in SCP-self-regulation, and that SCP-control recovers with increasing periods of alcohol abstinence.

METHOD

The same experimental procedures employed in Parts I and II of this publication series) were also used in the present investigation. Therefore apparatus and design will only be described briefly (for a more detailed description see Schneider, Rockstroh et al., 1992).

Subjects

Ten chronic alcohol-dependent male inpatients recruited from a 6-week detoxification program participated in this study. Only those subjects were accepted who met the diagnosis "alcohol dependency" according to DSM-III-R. Patients suffering from head trauma, migraine headache, stroke, other psychiatric illnesses (such as depression) or any other disease requiring medical treatment were excluded from the sample. Somatic withdrawal symptomatology had occurred recently and the patients were free of any withdrawal symptoms of the autonomic nervous system at study onset. Patients were all right-handed (Edinburgh Inventory, Oldfield, 1971). Table I provides the patient description.

Apparatus and Physiological Recordings

All aspects of experimental control and data acquisition were accomplished on-line by digital computers. Physiological signals were amplified with a 30-s time constant (high-frequency cutoff 30 Hz), digitized at 100 Hz, and digitally filtered to 10 points per second. EEG-recordings were taken from C_z referenced to linked earlobe electrodes separated from the common input by a resistor to prevent shunt currents between them. Vertical EOG recordings were measured between electrodes 1 cm above and 1 cm below the right eye.

Table I. Description and SCP Differentiation in the Second Transfer Block of the Patients with Alcohol Dependency

Patient	Age	Duration of alcohol dependency (in years)[a]	Alcohol consumption within the last 3 months (g/day)		Duration of abstinence before study onset (in days)	Gamma-glutamyl-transferase (I.U./1) at study onset	μV (average across session 1 to 4)
			Mean	Maximum			
1	28	9	165	420	4	34	0.03
2	30	3	200	420	0	13	−2.67
3	37	5	100	180	0	92	−2.01
4	34	6	185	330	0	155	−0.27
5	44	1	200	220	30	75	0.57
6	29	14	100	400	2	33	−2.10
7	39	5	180	220	12	75	2.87
8	31	1	160	200	0	106	−5.16
9	24	4	120	230	17	10	3.19
10	52	14	200	260	0	48	−3.39
Mean	35.2	6.2	161	287	6.5	64.1	−0.89

[a]Defined as the first loss of control and/or symptoms of somatic dependency.

Design and Procedure

Continuous visual SCP feedback was provided in each 8-s trial on a TV screen. The feedback stimulus was a rocket ship which moved in a horizontal plane through a gap. The subjects' task in each trial was to move the rocket ship from left to right out of the gap and to produce a supranormal SCP-shift on the "negativity" trial or a suppression of negativity on the "positivity" trial depending on the presented discriminative stimulus (either the letter A or B). At the end of each session the subjects received money on the bases of their success rate (between $2 and $8 (U.S.) per session).

The position of the rocket represented a linear function of the integrated EEG measured as change from the mean of a 1-s pretrial baseline. Whenever vertical eye movements of the same polarity as the required slow potentials were detected, a time-out contingency was used to prevent rocket movement (Elbert, Rockstroh, Lutzenberger, & Birbaumer, 1980).

"Transfer" trials were included in each session in order to assess response control without feedback (the rocket). These trials contained the letters A or B but no rocket. Each session consisted of 110 trials and was comprised of alternate blocks of 30 feedback trials and 20 transfer trials. Alcohol-dependent patients participated in 4 sessions on 4 consecutive days immediately following hospital admittance.

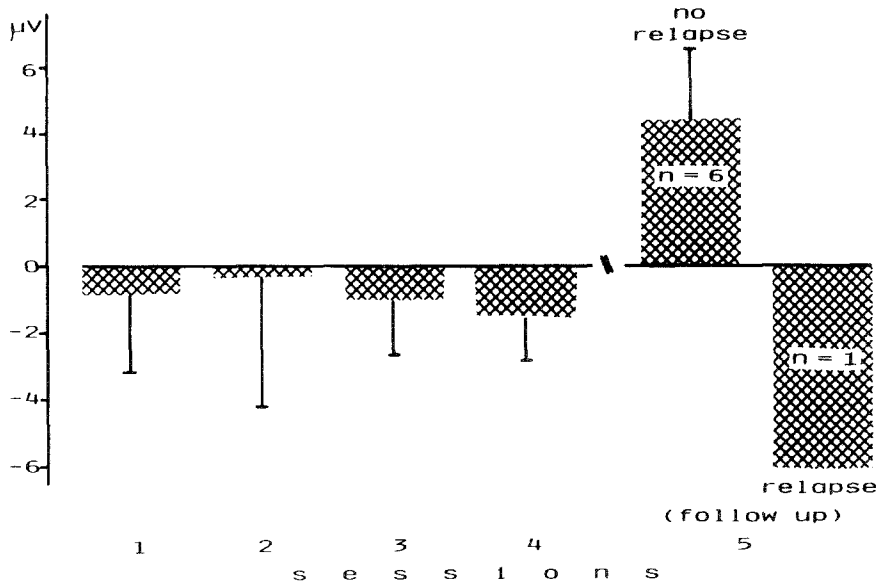

Fig. 1. Mean differentiation of SCPs in µV in the second transfer block between negativity and positivity trials in 10 patients with alcohol dependency over 5 sessions and 7 patients during the follow-up (bars mark µV averages).

Seven alcohol-dependent patients (Patient 1, 2, 3, 4, 5, 7, 10) agreed to participate in a fifth session some weeks after the detoxification treatment. During that time they participated in weekly group sessions in an outpatient treatment program. Abstinence was monitored with laboratory tests. Six of them stayed sober; one relapsed. Of the three remaining alcohol-dependent patients 2 were unable to attend because of their job and one canceled without explanation. The elapsed time between the 4th and 5th session was between 6 and 25 weeks which amounted to an average of 16.7 weeks (*SD* = 7.3).

Data Reduction and Analysis

Trials were excluded in which the DC-shift in the EEG-channel or EOG-channel exceeded 100 µV or 70 µV, respectively. Changes in vertical eye movements were assessed in the same manner as SCPs: There were no significant differences across the different conditions. SCPs were calculated by subtracting the mean SCP recorded during the last second of the

Table II. Mean Differentiation (+SE) Between Negativity and Negativity Suppression Trials in Microvolts

	Sessions 1-4[a]	Session 5 follow-up	
		No relapse[b]	Relapse[c]
Transfer 1	1.39	3.27	6.93
	±1.62	±2.33	
Feedback 1	0.70	2.28	−7.14
	±0.85	±2.78	
Feedback 2	3.90	2.38	−14.05
	±1.26	±2.24	
Transfer 2	−0.89	4.38	−5.60
	±0.85	±2.20	

[a] $n = 10$.
[b] $n = 6$.
[c] $n = 1$.

pretrial period from the mean SCP recorded during the 8-s feedback period.

Data from the last feedback-and-transfer trials of every session were submitted to ANOVAs and post-hoc tests.

RESULTS

SCP differentiations for the second transfer block across the 4 sessions for alcohol-dependent patients were not significant (−0.89 μV; $t(9) = -1.06$), in comparison to the results from the second feedback block (3.90 μV; $t(9) = 3.10$; $p = .006$) (Figure 1, Tables I and II).

At the follow-up (average of 4 months later) 6 alcohol-dependent patients (those without relapse) attained a significant SCP-differentiation of 4.38 μV ($t(5) = 1.99$; $p = .05$) during the second transfer block, but no significant differentiation during the second feedback block (2.38 μV; $t(5) = 1.06$). Patient 1 (who suffered a relapse within the follow-up period) was not able to successfully control his SCPs in the feedback or transfer trials.

DISCUSSION

At the beginning of the detoxification program alcohol-dependent patients were able to regulate their SCPs only in the presence of immediate feedback. Like schizophrenic patients at the beginning of their biofeedback training (Schneider, Rockstroh et al., 1992), alcohol-dependent patients were also unable to modify their SCPs in the transfer conditions; i.e., they were unable to generalize SCP control without the presence of immediate feedback.

Similar failures of SCP modification in SCP self-regulation tasks have been observed in subjects at risk for a psychotic development (Elbert, Lutzenberger, Rockstroh, & Birbaumer, 1983), in patients with bilateral frontal lesions (Lutzenberger et al., 1980), epileptic patients (Birbaumer, Elbert, Rockstroh, Daum, & Wolf, in press), and in individuals with attentional dysfunctions (Rockstroh, Elbert, Lutzenberger, & Birbaumer, 1990). Such similarities may indicate a common factor of impaired SCP regulation or, in patients, point to an impaired ability to adequately generalize SCP control. Signs of dysfunctional frontal lobe activity have frequently been observed in these patient groups. Since prefrontal regions are directly involved in the regulation of selective attention and the timing of behavior, particularly in delayed response tasks (Fuster, 1989), we assume that the regulatory deficit observed in patients with alcohol dependency and schizophrenia may reflect — at least in part — frontal lobe irregularities. This assumption is supported by findings that demonstrated that patients learned to perform responses upon command when immediate feedback and reward was provided, but completely failed to do so during transfer conditions, when reinforcement was delayed, i.e., was provided at the end of the session (monetary reward). This response pattern literally mimics the one that we observed earlier in patients with bilateral frontal lobe lesions (Lutzenberger et al., 1980).

In the literature frontocortical impairment in alcohol-dependent patients has been associated with attentional and behavioral disorders. Tarter (1975) reviewed the findings on alcohol-dependent patients and found that they match to a great extent findings of patients with frontal brain lesion. Skerchock and Cohen (1984) suggested that a reduced CNV in chronic alcohol-dependent patients indicates a specific frontal lobe dysfunction.

In the follow-up session (5th session) the effects of a longer abstinence period on SCP regulation were assessed. Six patients, who stayed sober for approximately 4 months, and 1 relapsed patient, participated in this session. SCP differentiation was significant in transfer for the patients without relapse and nonconsiderable for the relapsed patient. The results support the fact that the greatest SCP differentiation increase occurs during transfer. SCP differentiation in feedback was not significant, which may indicate that the information processing of the feedback stimulus may have prevented negativity development (Rockstroh et al., 1989). Thus, after a longer period of alcohol abstinence, patients learned SCP control as normals. This is supported by the present evidence, which shows that those patients abstinent more than 3 days before study onset already demonstrated positive differentiation during transfer in the first few sessions while all others achieved negative variations (see Table I).

In this study alcohol-dependent patients learned to modify their SCPs after abstaining from alcohol for a longer period of time. The hypothesis that the frontal lobe impairment in chronic alcohol-dependent patients is indeed reversible (Harper et al., 1988) is supported by our present findings. Our results are thus in agreement with reports that demonstrate evidence for reversibility of frontal lobe impairment, such as improvement in cognitive performance in alcohol abstinency after neuronal regeneration (McMullen, Saint-Cyr, & Carlen, 1984) and reversible brain damage (Carlen et al., 1978) even after extended periods of abuse.

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