Pain, 35 (1988) 205–213
Elsevier

PAI 01285

Biofeedback of somatosensory event-related potentials: can individual pain sensations be modified by biofeedback-induced self-control of event-related potentials?

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(Received 28 September 1987, revision received 11 May 1988, accepted 12 May 1988)

Summary This study investigates the effects of biofeedback based upon event-related brain potentials evoked by nociceptive electrical stimuli. In a visual and monetary feedback paradigm, 10 subjects received positive feedback within one training session when systematically showing two different behavior patterns: one pattern correlated with a decrease (down-training) and one with an increase (up-training) of the peak-to-peak size of the N150–P260 complex, respectively. Training conditions were changed randomly from trial to trial over 300 trials. All subjects achieved control on both behavior patterns resulting in a simultaneous modification of the size of this complex according to the training conditions. Furthermore, the individual pain report measured with a visual analogue scale was altered in accordance with the biofeedback-induced behavioral modifications. A decrease in subjective pain report was achieved after down-training while an increase was observed after the up-training.

Key words: ERP; Pain; Biofeedback; Electrical stimulation

Introduction

Several experimental studies have demonstrated that humans and animals may learn to control different cerebral activities by biofeedback procedures. However, whether these activities were modified directly or the feedback procedures affected complex behaviors of which the parameter used for the feedback procedure was only a part, has not been determined *.

In 1965, Olds reported an increase in the firing rate of tegmental cerebral neurons within mesen-

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* In most of these studies the term biofeedback was used interchangeably with other terms like operant or instrumental conditioning, or with terms referring to systems control theory (i.e., self-regulation, self-control, or self-modification). Because the problem of the underlying mechanisms of biofeedback is far from being resolved, we use here the term biofeedback as a metaphor for a procedure including feedback of physiological parameters for the purpose of affecting a systematic change in the quantity of the physiological process measured. Because one cannot separate electrical brain activities modified by feedback procedures from other behaviors involved, we refer to these electrical brain activities as behaviors.

cephalic and diencephalic areas in the rat [22]. Fetz and Finocchio [8] supported Olds' early findings in monkeys using external reinforcement to increase or decrease the firing rate of pyramidal tract and other cerebral neuronal units. Other studies in humans and animals have shown that spontaneous electrical CNS activities can be modified by biofeedback methods. Within this area of research, alpha-feedback has become the most common procedure, demonstrating that humans, like animals, may alter the activity within this frequency band by several methods of biofeedback [14,20]. Other studies have included operant control of activities within the theta frequency band [1,12,15], the sensorimotor rhythm [30], and high frequency band centered around 40 Hz [11,28,29].

Event-related potentials (ERPs) and slow brain potentials have been another well established area of biofeedback research, demonstrating that biofeedback induced changes of the amplitudes of visual, auditory and somatosensory electrical brain activities as well as changes of the polarity of slow brain potentials may lead to specific behavioral effects [7,24]. Only a few studies have investigated ERP biofeedback within a latency range of 800 msec. Finley [9,10] reported positive behavioral effects of biofeedback training, with quadriplegic subjects showing improved sensory functioning once the early components of cervical somatosensory evoked potentials (N14) were conditioned. In addition, he has shown that brain-stem auditory evoked potentials are amenable to operant control [9]. Roger [26] demonstrated biofeedback-induced modifications of the amplitudes of visual ERPs. Miltner et al. [16] investigated an operant control paradigm to modify the P300 of the visual ERP and showed that an increase in the positivity of P300 was associated with a decrease in reaction time. Most of the work on the operant control of somatosensory event-related potentials within the latency range up to 500 msec was done by Rosenfeld and his group [5,6,27]. This work shows that both humans and animals may regulate the amplitude of nociception-related ERPs leading to changes in the perception of pain.

In dolorimetric studies using somatosensory ERPs as objective parameters of pain perception, many research groups have substantiated that the N150–P260 complex of the ERP is very sensitive to different intensities of nociceptive stimuli. The size of this complex correlates with the subjective pain report [2,3,19]. Furthermore, analgesics reduce the amplitudes of this complex as well as the verbal pain report [3].

The present study investigated whether human subjects are able to modify the peak-to-peak size of the N150–P260 complex of the ERP by using a visual and monetary biofeedback paradigm. Furthermore it investigated whether a change of the N150–P260 complex is associated with a concurrent modification of pain perception.

Methods

Subjects

Ten male healthy volunteers aged between 21 and 46 participated as paid subjects. Subjects were informed about the course of the experiment and gave written informed consent according to the Helsinki convention. No subject was under current medication and no one suffered from any disease.

Procedure

For each subject, the experiment consisted of 1 session lasting about 2 h. During the first part of the session, both the individual pain threshold (ITP) and responses to a stimulus with an intensity of 20% above the individual pain threshold (ITP20) were measured. During a second part of the session, the baseline ERPs and subjective pain reports according to ITP20 were recorded. The last part of the session was devoted to the feedback training of the electrical brain response. In order to elicit the ERP to be modified, a stimulus intensity of ITP20 was used again (see Table I).

Pain threshold measurement

A standardized method of intracutaneous electrical stimulation was employed [2a,17], using weak electrical currents with an intensity of between 70 and 900 μA applied to the subcutaneous layers of a finger tip of the non-dominant hand. The anode was an isolated golden pin of 1 mm length and 0.9

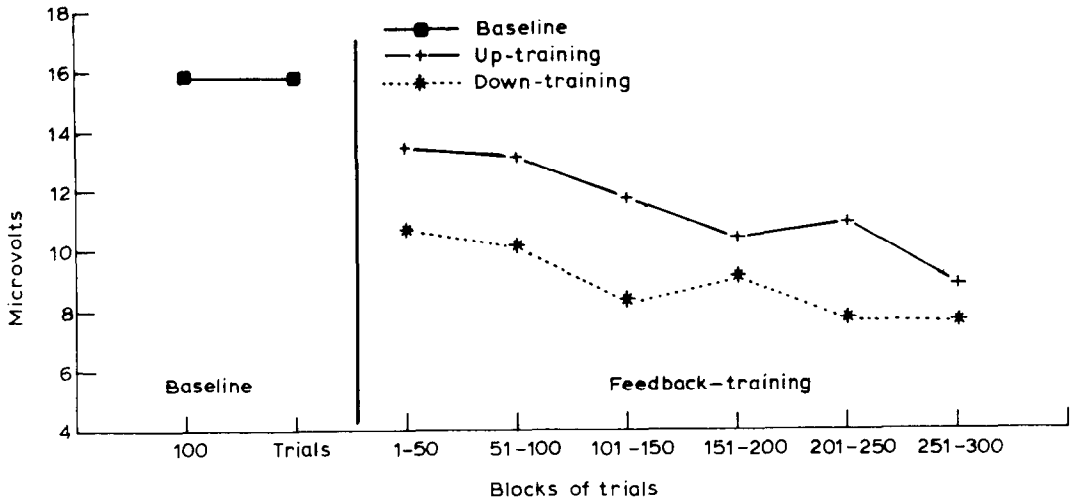

Fig. 1. Figure displays the effects of a single session feedback training of the peak-to-peak size of the N150–P260 complex of single trial SEPs elicited by an intracutaneous electrical stimulus of 10 msec duration. Mean peak-to-peak sizes of the N150–P260 amplitude during pre-training baseline and the mean N150–P260 amplitudes averaged for 6 blocks of 50 successive trials during training are displayed. Solid line shows the effects of up-training, dotted line represents the down-training condition. Up- and down-training conditions were changed randomly from trial to trial (see text).

mm diameter inserted into a prepared epidermal cavity and properly fixed. The cathode consisted of a flexible stainless steel electrode worn by the subject on the same finger. Preparation of the cavity was carried out carefully to prevent bleeding, but was sufficiently deep to ensure that the skin resistance fell below 10 kΩ. Subjects were grounded with a flexible band-like and moist electrode fixed around the wrist.

This intracutaneous stimulation technique has the advantages over other methods (i.e., superficial stimulation) that subjects perceive intracutaneous stimulations as being sharp, distinct and easy to localize; secondly, the current power needed for high intensity stimuli is reduced by a factor of about 10 due to a reduced skin resistance, thus minimizing electrical artifacts in the physiological measurements; thirdly reliability of the intracutaneous stimulation is high between different sessions and within a single long lasting session [2a,17].

TABLE I

Part 1	Part 2	Part 3
Experimental design		
Determination of individual pain threshold	Baseline Variation of ERP and VAS	Biofeedback Up-training Down-training
Stimulus intensity		
Varied	Constant ITP20	Constant ITP20

The intracutaneous electrical stimulus (IES) was delivered by a programmable constant current stimulator linked to the subject via an isolation unit supplying a maximum voltage of 150 V and a maximum current of 1 mA. Current was delivered by a 16 V battery.

The IES consisted of a bipolar rectangular pulse of a duration of 10 msec totally. During part 1 (individual pain threshold measurements), subjects received 2 consecutive series of stimuli, each of about 40 IES. Each series started with an intensity of zero and increased stepwise to a maximal intensity of about 60% above individual pain threshold. Then the intensity was decreased stepwise back to zero. Each IES was indicated by a visual warning stimulus presented on a video screen 1000 msec prior to its occurrence. The warning stimulus consisted of a light square of 24.4 cm^2. The interstimulus interval of IES was randomized varying between 9 and 12 sec. Three seconds after presentation of each stimulus sub-

jects were asked to rate the subjective intensity of the stimulus by using a visual analogue scale (VAS) presented on a video screen placed 3 m in front of the subject (see Fig. 1). Its height could be increased by pressing a microswitch. The VAS contained 3 lines, one at the bottom of the scale indicating 'no sensation,' one 10% below the midline 'just perceivable pain' and one at the top of the scale 'unbearable pain.' Actual numbers of IES in this part of the experiment depended on the number of trials until the intensity of 20% below the threshold for 'unbearable pain' was reached. From this stimulus level decreasing intensities were applied stepwise until subjects reported no sensation. This increasing and decreasing stepwise procedure was repeated once. The final individual pain threshold was calculated by averaging the intensities of all 4 pain thresholds reported within both runs. A stimulus of 20% above pain threshold was applied as the nociceptive stimulus (ITP20) in all subsequent stages of the experiment.

Pre-training baseline

During this part of the experiment, 100 IES with the ITP20 intensity were applied to each subject. The method of stimulation (warning signal and IES), the time course of one trial and the intertrial intervals were identical to those of the first part of the experiment. Again the VAS was presented 3 sec after each single IES and the subjective pain report was taken after each block of 10 trials. Furthermore, several physiological parameters were recorded before and after stimulation, including EEG, EOG and EMG. Artifact-free EEGs of this period were averaged for the mean peak-to-peak size of the N150–P260 complex of ERP from each subject, serving as reference values for the amount of reinforcement given to each subject during training.

Feedback training

The following feedback training was composed of a series of 300 IES identical to those of the pre-training baseline using the ITP20. Again each IES was indicated by a visual warning signal presented 1 sec before IES in the middle of a video monitor. The warning signal during feedback training was either a symbolized male or female figure.

Two different tasks were indicated by this warning stimulus: one in which the subjects were requested to increase the peak-to-peak size of the N150–P260 complex of ERP (up-training) and one in which they should decrease this complex (down-training). Presentation of both warning stimuli was randomized within trials. Thus the task conditions (i.e., up- and down-training) were changed randomly from trial to trial. Subjects were not informed about the relationship between style of figures and the kind of task. They were only instructed to fixate the warning figure. After the application and perception of the IES (ITP20), their task was to produce as many figures as possible on the video screen. After presentation of a male warning signal, subjects were asked to increase the number of male figures; after a female warning signal the task was to increase the female figures. Male and female figures were related to either up-training or down-training in a balanced order among subjects.

The number of figures displayed was computed by dividing the actual peak-to-peak size of the N150–P260 complex by the mean peak-to-peak size of the individual pre-training baseline. During up-training trials large positive deviations of the actual single trial peak-to-peak size from this baseline value resulted in a large number of figures; when the actual size was smaller than the baseline peak-to-peak size no feedback was given. During down-training the number of figures displayed depended on to what extent the actual size of the N150–P260 complex fell below the baseline value. Actual positive or negative deviations of more than 1 S.D. from the baseline mean resulted in 3 figures (either male or female figures depending on the task condition) on the screen. The maximum number of male or female figures was 11; the minimum was 1. To prevent mediation by eyeblinks or vertical eye movements feedback was only given when the trial was not contaminated by any eye movements. In addition, after each non-contaminated trial a monetary bonus was displayed at the top of the video screen. When the subjects showed too much eye blinks or movements during the feedback trials, they were re-

quested not to blink or to move their eyes between the warning stimulus and the end of the trial.

After each block of 50 trials the VAS was displayed twice and the subject was requested to rate the intensity of the mean pain sensations experienced during both the up- and down-training conditions respectively.

Physiological recordings

The EEG was recorded from vertex (Cz) using an Ag/AgCl electrode (In Vivo Metrics). An earclip fixed to the right ear served as reference. Eyeblinks and eye movements were controlled by a vertical electrooculogram (EOG) recorded from 1 cm above and below the midline of the left eye using Beckman Ag/AgCl electrodes. Electromyographical responses (EMG) due to the pain stimulus onset were measured from the digital extensor muscle, 5 cm above the wrist.

The physiological recordings were monitored by a Beckman Dynograph Type R using Beckman 9806A couplers for EEG and EOG. A Model 9852 served as a coupler for the EMG recordings. The time constant of the EEG and EOG coupler was extended to 5 sec, the high frequency cut-off filter was set at 30 Hz.

A DEC PDP 11/73 computer controlled the timing and display of all experimental stimuli and the registration of the individual responses. Within one trial the EEG, EOG, and EMG responses were digitized on-line during the first 3 sec of each trial using a DEC ADF01 interface set at a conversion rate of 100 Hz. Data were stored trial by trial on a hard disk for further off-line analysis.

After initial information, subjects sat in a reclining chair in an electrically shielded, dimly lit, and sound-attenuated room. Instructions were given verbally and in written form via the video monitor. Then subjects were requested to sign the informed consent document. Electrodes were attached and subjects were asked to relax. At the end of the experiment each subject was paid for participation and received the amount of bonus achieved during training.

Data reduction and analysis

Prior to analysis both EOG and EMG artifact-contaminated trials were completely discarded from the database when the intensity of EOG exceeded 20 μV and that of the EMG 5 μV. This procedure resulted in an average exclusion of 18% of all trials across subjects.

The remaining artifact-free trials of each subject were averaged according to the up- or down-training condition for 6 blocks of 50 consecutive artifact-free single trials resulting in a set of 12 EEG traces per subject. Next the peak-to-peak size of the averaged N150–P260 complex of the ERP of each EEG trace was extracted using a computerized peak detection algorithm. The window for the N150–P260 complex was set from 100 to 500 msec. These 36 data sets of each subject were then submitted to an analysis of variance (ANOVA) using a model for repeated measurements with the within-subject factors BLOCKS (blocks 1–6) and TRAINING CONDITION (up-/down-training).

The 12 subjective pain reports of each subject were pooled according to both training conditions and submitted to an analysis of variance for repeated measurements again with the within-subject factors BLOCKS and TRAINING CONDITION.

Results

Fig. 1 and Table II summarize the means and standard deviations of the peak-to-peak N150–P260 amplitudes according to the 6 blocks of trials and the 2 feedback-training conditions. There was a significant difference in the size of the N150–P260 complex between the up- and down-training condition (F (1, 9) = 10.42; $P < 0.01$). Furthermore, there was a systematic decrease in the size of this complex from block 1 to block 6 independent of the training condition, indicating habituation (F (5, 45) = 7.03, $P < 0.01$). Because up- and down-training conditions were changed randomly from trial to trial during the course of the experiment, N150–P260 amplitudes of both conditions were affected equally by this habituation process. This habituation effect is supported by the lack of a significant interaction between the factors BLOCKS and TRAINING CONDITION (F (5, 45) = 1.18, $P = 0.33$). Feedback results are,

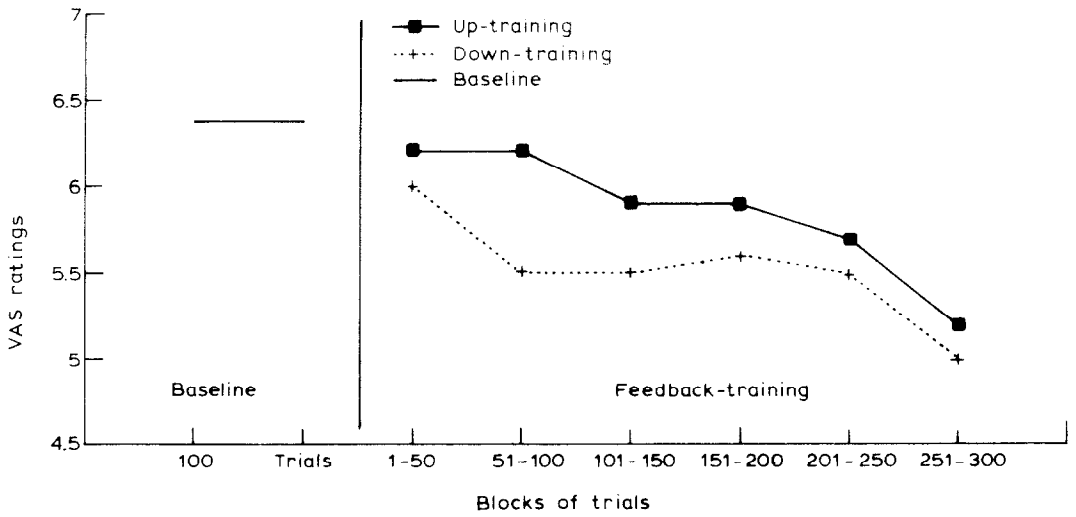

Fig. 2. Figure shows the effects of the feedback training on the subjective pain report. Arrangement of lines follows that used in Fig. 1. For further details, see text.

therefore, not explainable in terms of habituation but represent a unique source of variance. Furthermore, as Fig. 1 indicates, the acquisition of self-control can be achieved very rapidly.

Similar to the ERP changes, the subjective pain reports show significant differences between up- and down-training (Fig. 2 and Table III). As the analysis of the VAS measurements indicate, the

TABLE II

MEAN PEAK-TO-PEAK SIZE AND STANDARD DEVIATION OF N150–P260 COMPLEX OF SEP DURING 6 CONSECUTIVE BLOCKS OF BIOFEEDBACK TRAINING RELATED TO UP- AND DOWN-TRAINING CONDITIONS

Block	Trials	Up-training (μV)		Down-training (μV)	
		Mean	S.D.	Mean	S.D.
1	1– 50	13.39	3.28	10.64	4.04
2	51–100	13.10	4.23	10.08	3.41
3	101–150	11.70	3.37	8.21	3.12
4	151–200	10.37	2.70	9.04	2.78
5	201–250	10.90	3.48	7.64	2.97
6	251–300	8.82	2.83	7.58	2.85

Analysis of variance:

Factor	Degrees of freedom	F value	P
Blocks	(5, 45)	7.03	0.001 ss
Training conditions	(1, 9)	10.42	0.0104 ss
Blocks × training conditions	(5, 45)	1.18	0.3321 ns

TABLE III

CHANGES OF SUBJECTIVE PAIN REPORT

Mean pain ratings (VAS) and standard deviations at the end of 6 blocks related to up- and down-training conditions. VAS ranged from 0 (no sensation), 4 (pain threshold) to 8 (unbearable pain).

Block	End of trial	Up-training		Down-training	
		Mean	S.D.	Mean	S.D.
1	50	6.2	0.82	6.0	0.71
2	100	6.2	1.10	5.5	0.75
3	150	5.9	1.16	5.5	1.18
4	200	5.9	1.37	5.6	1.16
5	250	5.7	1.41	5.5	1.43
6	300	5.2	1.63	5.0	1.62

Analysis of variance:

Factor	Degrees of freedom	F value	P
Blocks	(5, 45)	2.23	0.07 ns
Training conditions	(1, 9)	5.71	0.04 s
Blocks × training conditions	(5, 45)	1.45	0.22 ns

up-training trial stimuli were rated as being more painful than were those of down-training trials ($F(1, 9) = 5.71$, $P < 0.05$). In contrast to the ERPs the subjective pain report did not show habituation, i.e., there was no significant decrease in the ratings with time, independent of the training conditions.

As indicated in Fig. 1, subjects were unable to increase the N150–P260 complex during the up-training trials beyond the baseline level. The N150–P260 complex remained smaller in the two training conditions compared to baseline condition. Training effects of the two conditions cannot, therefore, be considered in relation to baseline but only in relation to each other. This comparison shows that up-training trials resulted in larger N150–P260 amplitudes than do down-training trials.

Interviews at the end of the training session revealed that subjects used a broad spectrum of different cognitive strategies to comply with the feedback tasks. During the up-training condition, some subjects reported concentrating on the stimuli applied, whereas others tried to relax or imagine positive interactions with others. During the down-training condition, similarly varying reports were given. Some subjects did not realize that their brain activities were being used as the basic parameter for the feedback procedure.

Discussion

The present study confirms earlier findings by Rosenfeld and coworkers [5,6,27], demonstrating that ERPs may be brought under biofeedback control in humans. In contrast to Rosenfeld's studies where subnoxious evoking stimuli were used in our study painful stimuli were applied. Also the feedback procedure was different. This may account for the fact that our subjects were more rapidly able to control the ERP amplitude than Rosenfeld's subjects. Like the differential effects of the up- and down-training conditions on the size of the N150–P260 complex of ERP, the subjective pain report was increased under the up-training and decreased under the down-training condition.

Although this effect was rather small, the outcome emphasizes the strong relationship between the size of the N150–P260 complex and the subjective pain experience as found in many previous studies [2–4,19].

Whether the results reflect an operant conditioning, a control system process [20], or a cognitively mediated control process whereby the subjects were able to control the electrical brain activities cannot be determined by our data. However, since our subjects used rather different cognitions about the control of electrical brain activity it can be concluded that: (1) there is no particular cognitive strategy related to successful control, and (2) that cognitions may be irrelevant to control. An operant learning process or a systems control explanation would seem better suited to this modification.

As one can see in Fig. 1, the peak-to-peak size of the N150–P260 complex was usually smaller under both biofeedback conditions compared to baseline. The interviews at the end of the session revealed that it was difficult for all subjects during the up-training trials to behave consistently in such a way that positive feedback was achieved. According to the experimental design, positive feedback during this condition was related to the individual's ability to increase the actual N150–P260 peak-to-peak size above the baseline level of this complex. It is uncertain whether it was more difficult for subjects to increase or to decrease this N150–P260 complex. Under the up-training condition the increase must overcome an opposing process of habituation resulting in a decrease in the ERP amplitudes. Consequently, all subjects may become demotivated after a number of trials of the up-training task, and then primarily concentrate on the down-training task. We further speculate that our subjects did not want to exceed the up-training brain activities beyond the baseline level because they did not want to experience more pain. In the context of operant learning this reflects negatively reinforced avoidance behavior. In contrast, the down-training effects may be the result of both a positive (monetary bonus) and a negative reinforcement (avoidance or pain) process, respectively.

A third explanation for the difficulty in extend-

ing the baseline is that during baseline, subjects had to perform one single task (perception and responding to one stimulus), whereas during feedback tasks were more complex (differentiating 2 warning stimuli indicating different tasks, processing the painful stimulus and finally preparing and complying with the requested response). A number of studies have shown that attention to a single stimulus generally results in an increase in the power of the resulting ERP amplitudes. In contrast, splitting attention to different task relevant stimuli and being engaged in difficult preparational processes simultaneously leads to a decrease in ERP amplitudes [13,18,21].

Despite these limitations in achieving control over a behavior related to different electrical brain activities within a rather small latency window (N150–P260), our study significantly demonstrated that humans are able to control their pain experience by means of an ERP feedback paradigm. Broadly, our data support the idea that the N150–P260 size does not solely reflect neurophysiological aspects of stimulus processing but rather complex psychological aspects, i.e., attentional aspects, cognition, processing of stimulus information and response preparation.

Replications of this study and further laboratory research with acute or chronic pain patients are required to determine whether these findings have any significance for clinical procedures of pain control.

Acknowledgements

This research was supported by an SFB Grant No. SFB 307/B2, awarded to Wolfgang Miltner by the Deutsche Forschungsgemeinschaft, Bonn.

We would like to thank Drs. N. Birbaumer, T. Elbert, B. Rockstroh, and W. Lutzenberger for valuable suggestions and technical assistance.

References

1 Beatty, J., Learned regulation of alpha and theta frequency activity in the human brain. In: G. Schwartz and J. Beatty (Eds.), Biofeedback, Theory and Research, Academic Press, New York, 1977, pp. 351–370.

2 Bromm, B., Pain-related components in the cerebral potential. Experimental and multivariate statistical approaches. In: B. Bromm (Ed.), Pain Measurement in Man. Neurophysiological Correlates of Pain, Elsevier, Amsterdam, 1984, pp. 257–290.

2a Bromm, B. and Meier, E., The intracutaneous stimulus: a new pain model for algesimetric studies, Meth. Find. Exp. Clin. Pharmacol., 6 (1984) 405–410.

3 Chapman, C.R. and Jacobson, R.C., Assessment of analgesic states: can evoked potentials play a role. In: B. Bromm (Ed.), Pain Measurement in Man. Neurophysiological Correlates of Pain, Elsevier, Amsterdam, 1984, pp. 233–255.

4 Chudler, E.H. and Dong, W.K., The assessment of pain by cerebral evoked potentials, Pain, 16 (1983) 221–244.

5 Dowman, R. and Rosenfeld, J.P., Operant conditioning of somatosensory evoked potential (SEP) amplitude in rats. I. Specific changes in SEP amplitude and a naloxone-reversible somatotopically specific change in facial nociception, Brain Res., 333 (1985) 201–212.

6 Dowman, R.J., Rosenfeld, J.P. and Heinricher, M., Operant conditioning of trigeminally evoked cortical potentials: correlated effect on facial nociception, Brain Res., 269 (1983) 111–118.

7 Elbert, T. and Rockstroh, B., Classification and overview of CNS electrical activity tested on operant conditioning. In: T. Elbert, B. Rockstroh, W. Lutzenberger and N. Birbaumer (Eds.), Self-Regulation of the Brain and Behavior, Springer, Heidelberg, 1984, pp. 59–63.

8 Fetz, E.E. and Finocchio, D.V., Correlations between activity of motor cortex cells and arm muscles during operantly conditioned response patterns, Exp. Brain Res., 23 (1975) 217–240.

9 Finley, W.W., Biofeedback of very early potentials from the brain stem. In: T. Elbert, B. Rockstroh, W. Lutzenberger and N. Birbaumer (Eds.), Self-Regulation of the Brain and Behavior, Springer, Heidelberg, 1984, pp. 143–163.

10 Finley, W.W. and Johnson, G., Biofeedback of brainstem auditory evoked potentials. In: Proc. 12th Annual Meeting of the Biofeedback Society of America, 1981, pp. 15–18.

11 Ford, M., Bird, B.L., Newton, F.A. and Sheer, D., Maintenance and generalization of 40 Hz EEG biofeedback effects, Biofeedback Self-Regul., 5 (1980) 193–205.

12 Hauri, P., Biofeedback techniques in the treatment of chronic insomnia. In: R. Williams and I. Karacan (Eds.), Sleep Disorders: Diagnosis and Treatment, Wiley, New York, 1980.

13 Hillyard, S.A., Event-related potentials and selective attention. In: E. Donchin (Ed.), Cognitive Psychophysiology: Event-Related Potentials and the Study of Cognition (The Carmel Conferences, Vol. 1), Lawrence Erlbaum, Hillsdale, NJ, 1984, pp. 51–72.

14 Kamiya, J., On the relationships among subjective experience, behavior, and physiological activity in biofeedback learning. In: T. Elbert, B. Rockstroh, W. Lutzenberger and N. Birbaumer (Eds.), Self-Regulation of the Brain and Behavior, Springer, Heidelberg, 1984, pp. 245–254.

15 Lutzenberger, W., Birbaumer, N. and Wildgruber, C., An experiment on the feedback of the theta activity of the human EEG, Eur. J. Behav. Anal. Modific., 2 (1975) 119–126.
16 Miltner, W., Larbig, W. and Braun, C., Biofeedback of visual evoked potentials, Int. J. Neurosci., 29 (1986) 291–303.
17 Miltner, W., Larbig, W. and Braun, C., Tooth pulp and intracutaneous electrical stimulation. Major properties and differences, Unpublished manuscript, University of Tuebingen, 1987.
18 Miltner, W., Larbig, W. and Braun, C., Arousal, attention and event related potentials elicited by intracutaneous electrical stimulation of the skin, J. Psychophysiol., 2 (1988) in press.
19 Miltner, W., Larbig, W. and Braun, C., Habituation of subjective ratings and event related potentials to painful intracutaneous electrical stimulation of the skin, J. Psychophysiol., 2 (1988) in press.
20 Mullholland, T.B., Concepts of control in biofeedback. In: T. Elbert, B. Rockstroh, W. Lutzenberger and N. Birbaumer (Eds.), Self-Regulation of the Brain and Behavior, Springer, Heidelberg, 1984, pp. 277–295.
21 Näätänen, R., Gaillard, A.W.K. and Mäntysalo, S., Brain potential correlates of voluntary and involuntary attention. In: H.H. Kornhuber and L. Deecke (Eds.), Motivation, Motor, and Sensory Processes of the Brain. Electrical Potentials, Behavior, and Clinical Use, Elsevier, Amsterdam, 1980, pp. 343–348.
22 Olds, J., Operant Conditioning of Single Unit Responses, Excerpta Medica, Amsterdam, 1965, pp. 372–380.
23 Rhodes, D.L., Buffington, V. and Chapman, C.R., Effects of attention/distraction on evoked potentials to painful and non-painful gingival stimulation, Pain, Suppl. 4 (1987) S183.
24 Rockstroh, B., Elbert, T., Birbaumer, N. and Lutzenberger, W., Slow Brain Potentials and Behavior, Urban and Schwarzenberg, Munich, 1982.
25 Rösler, F., Claussen, M. and Sojka, B., The double-priming paradigm: a tool for analyzing the functional significance of endogenous event-related brain potentials, Biol. Psychol., 22 (1986) 239–268.
26 Roger, M., Operant control of evoked potentials: some comments on the learning characteristics in man and on the conditioning of subcortical responses in the curarized rat. In: T. Elbert, B. Rockstroh, W. Lutzenberger and N. Birbaumer (Eds.), Self-Regulation of the Brain and Behavior, Springer, Heidelberg, 1984, pp. 180–197.
27 Rosenfeld, J.P., Dowman, R., Silvia, R. and Heinricher, M., Operant controlled somatosensory brain potentials: specific effects on pain processes. In: T. Elbert, B. Rockstroh, W. Lutzenberger and N. Birbaumer (Eds.), Self-Regulation of the Brain and Behavior, Springer, Heidelberg, 1984, pp. 164–179.
28 Sheer, D.E., Biofeedback training of 40 Hz EEG and behavior. In: N. Barch and H.L. Altschuler (Eds.), Behavior and Brain Electrical Activity, Plenum Press, New York, 1975.
29 Sheer, D.E., Focused arousal, 40-Hz EEG, and dysfunction. In: T. Elbert, B. Rockstroh, W. Lutzenberger and N. Birbaumer (Eds.), Self-Regulation of the Brain and Behavior, Springer, Heidelberg, 1984, pp. 64–84.
30 Sterman, M.B., The role of sensorimotor rhythmic EEG activity in the etiology and treatment of generalized motor seizures. In: T. Elbert, B. Rockstroh, W. Lutzenberger and N. Birbaumer (Eds.), Self-Regulation of the Brain and Behavior, Springer, Heidelberg, 1984, pp. 95–106.