

## **Effects of different Biofeedback training procedures on quantitative Electroencephalographic parameters of healthy subjects**

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# *Table of contents*

Introduction	4
Methods	14
Participants	14
Material	14
Design and procedure	14
Physiological recordings	15
Pre- and Post QEEG	17
Data analysis and reduction	18
Results	20
Analysis of Pre- and Post QEEG	20
Analysis of behavioral data	21
Analysis of physiological data	25
Correlation	30
Discussion	32
References	36
Appendix 1	38
Appendix 2	40
Appendix 3	41
Appendix 4	42

## ***Introduction***

Epilepsy is a disease which leads to disturbances of multiple functions and which can cause changes in behaviour. It is a condition which is characterized by excessive and abnormal activity of the brain. This abnormal activity can manifest itself in different ways. Sometimes a patient completely loses his consciousness and lies on the ground shaking. For others, it only leads to a subtle movement in the face. An insult can be observed in the EEG as large amplitude oscillations. An epileptic insult can be restrained to a small part of the brain (focal seizures) or the whole brain can be involved (generalized seizures). A seizure can be simple, when there is no loss of consciousness, or it can be complex when there *is* loss of consciousness.

Throughout the years many theories have been developed about possible origins of epilepsy. Furthermore, by means of research, knowledge has been obtained about the possible mechanisms underlying seizures. It is generally accepted that a seizure is a disturbance in cortical excitability. During a seizure there is overexcitation of neurons. (Niedermeyer & Lopes de Silva, 1999)

The existence of two opponently functioning neurotransmitter systems is part of the balance that keeps the brain functioning in a healthy way. The level of excitability of a neuron at a certain moment in time is the result of competing influences of depolarization and hyperpolarization. Normally these processes are balanced; excitatory neurotransmitters *facilitate* excitatory bursts while inhibitory neurotransmitters *inhibit* action potentials. In case of epileptic activity this balance is disturbed. Various processes have been found to be involved in the occurrence of an action potential. A neuron is interconnected with different sorts of neurons in the central nervous system (CNS) and receives its information from other neurons by a neurochemical exchange of neurotransmitters. There exist two different classes of neurotransmitters; excitatory and inhibitory neurotransmitters. When these are secreted at the synapse, they either excite or inhibit other neurons that are contacted through this synapse. Within a neuron there is a different concentration of positive and negative ions on the inner and outer side of the cell-membrane. Electrophysiological recordings of the resting potential shows that the inside of the cell has a negative charge that is about  $-70$  mV. When a neuron is stimulated at the dendrites two successive processes take place; sodium ions flow into the cell which cause the resting potential to increase. This means depolarization of the cell, which is

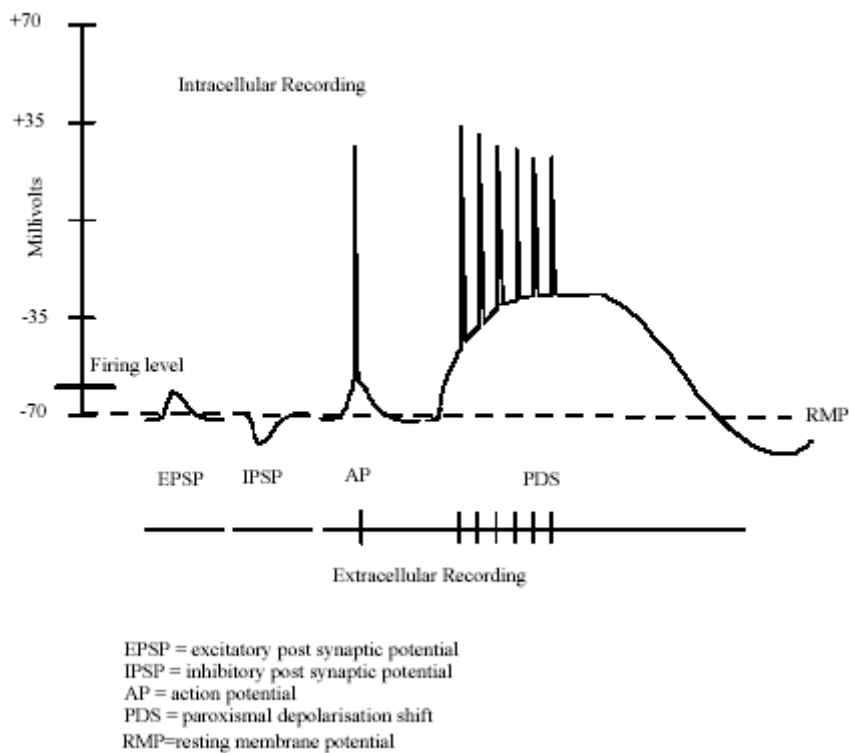
called an excitatory postsynaptic potential (EPSP). The second phase is an inhibitory potential which has a longer latency period. This hyperpolarization is called an inhibitory postsynaptic potential (IPSP). This process is a consequence of increased permeability of the cell membrane caused by potassium and chloride influx. (Kandel et al, in principles of neural science)

The EPSPs and IPSPs will be summated. Consequently, if a stimulus is sufficiently intense and prolonged, the dendritic potential reaches the firing threshold of approximately  $-60$  mV. This causes a huge depolarization and the membrane potential increases to  $+35$  mV. This firing is called an action potential.

After propagation of the action potential over the axon of the neuron it arrives at the synapse. There, a secretion of a neurotransmitter will take place. This will lead to either excitation or inhibition of postsynaptic neurons. Normally, the generation of an action potential takes a couple of milliseconds. If the contribution of EPSPs is exaggerated or the occurrence of IPSPs is inhibited, this will result in consecutive action potentials. This is called a paroxysmal depolarization shift (PDS).

Two main excitatory neurotransmitters, glutamate and aspartate, have an excessive contribution in the synaptic cleft and other extracellular spaces. These have a main role in initiation, spreading and course of epileptic activity. Gamma amino butyric acid (GABA) is the main inhibitory neurotransmitter in the brain. This neurotransmitter has an inhibitory effect on neuronal activity. In the case of epileptic activity there is secondary to excessive presence of excitatory neurotransmitters, a deficiency in inhibitory neurotransmitters.

A seizure starts at a certain location in the brain and spreads to surrounding areas. The initial phase of a seizure is characterized by the two following events. First, there are high-frequency discharges of action potentials. Thereafter, a hypersynchronization of a neuronal population occurs. These synchronized discharges from the neuronal population can be seen as spike-discharges in the EEG. Focusing on individual neurons, epileptic activity is neuronal depolarization that results in a burst of action potentials, a plateau-like depolarization after the end of the burst. Depolarization is followed by repolarization and hyperpolarization. This is the course of a PDS.



*Fig 1: Time-course of a paroxysmal depolarization shift*

The majority of epileptic patients benefits from medication, which is often based on GABA-ergic drugs, an inhibitory neurotransmitter, preventing the seizure activity to initiate and spread. However, about 30% of patients respond insufficiently to medication or have an epileptic focus that is not suitable for neurosurgery. Besides this, the fact that anti-epileptic drugs do not cure (but only suppress seizures), indicates that current treatment of epilepsy is not satisfactory. Therefore, it is important to search for alternative treatments for epilepsy. In this search there has been a growing interest in Neurofeedback, or EEG-Biofeedback, as a treatment for epilepsy during the last years. Neurofeedback is a type of biofeedback. Biofeedback is effective by means of visualizing unconscious psycho-physiological processes, in a way that people can exert voluntary control over their physiology.

Neurofeedback is biofeedback of the EEG. This means that when a person receives information about what is happening in his brain, the processes in the brain can be affected. Neurofeedback is based on two principles. Electric activity, as is measured by the EEG, reflects neurophysiological processes in the brain. The second principle is that processes in the brain can be altered by training or learning. In a neurofeedback training a person is

instructed to modulate specific frequency patterns of his EEG. As a person changes these patterns he influences his brain processes. The EEG is transformed in a pleasant representation on the computer screen. Regulation of the representation on the computer screen reflects self-regulation of the EEG. This self-regulatory skill means that underlying psycho-physiological processes are affected.

The main process by which this self-regulatory skill can be achieved is by means of operant (or instrumental) conditioning. From learning psychology it is known that a consequence of rewarding a certain behaviour is an increased probability of that behaviour. (Thorndike's Law of effect) The probability of a certain behaviour is strengthened when this is followed by a positive consequence (reinforcer). In neurofeedback, when reinforcement of the behavior is desirable, there are three important conditions; the reinforcer must follow the response, the reinforcer must follow immediately and the reinforcer must be contingent on the response. (Liebermann, learning behavior and cognition). In neurofeedback the reinforcer generally is the advancement in a film, a sound or a visual display. The reinforcer is contingent to the occurrence of an EEG pattern, which is considered desirable for the treatment.

Research indicated neurofeedback is a very promising treatment for epilepsy. The beneficiary effects for epileptics were first discovered by Sterman et al. (1972). Sterman et al. studied the sensorimotor rhythm (SMR) The SMR is a rhythm in the frequency range of 12-15 Hz which is located over the sensory motor cortex. (C3, CZ and C4) Sterman (1967) did his early experimentation on ten cats that were conditioned to produce this brainwave. Some years later an experiment was performed for NASA to examine the toxic effect of rocket fuel. There was an indication that this substance was very neurotoxic and caused epileptic seizures. In this study Sterman used fifty cats. All cats were injected with the fuel and after an hour almost every cat showed severe seizures and eventually died. An exception, however, were the SMR-trained cats that also participated in the previous experiment. Seven of these SMR trained cats displayed significant delay in seizure onset, whereas three did not have a seizure at all. Apparently, the SMR training had raised their seizure threshold. This was the first indicator of the promising effect of neurofeedback for epileptics. However, it took four years before the first anti-epileptic effects on a human being were reported (Sterman, 1971) There was a 23 year old female patient who had drug-refractory frontoparietal epilepsy. She was trained to raise her sensorimotor rhythm (SMR) which resulted in a decrease of epileptic seizures. From this point on there has been ongoing interest and research into the anti-epileptic effects of

neurofeedback.

The SMR neurofeedback protocol for epileptics is extensively studied since publication of Sterman et al (1971). People can learn to increase the SMR by operant conditioning. It turned out that an increase in the SMR has an anti-epileptic effect. This indicates that an increase in SMR normalizes the EEG. The first studies into SMR training that were published have been severely criticized because of poor experimental control and poor description of epileptic patients. Nowadays better research has been done. As a result of this research the mechanisms of SMR neurofeedback training are well understood and the training is a reliable treatment. In a review of Sterman (2000) a meta-analysis was performed. It turned out that out of 174 patients 82% reached a significant seizure-reduction (more than 30%). It was concluded that most epileptic patients who show clinical improvement also show contingency-related EEG changes and their EEG was more normalized.

A second neurofeedback method that is advantageous for epileptics is slow cortical potential training. Slow cortical potentials (SCPs) are potential changes in the cerebral cortex which are in the frequency domain of 0-2 Hz and have a duration of several seconds. SCPs are not specifically localized and therefore are a manifestation of common activity of different neuronal regions. (McCallum Slow potential changes in the human brain, 1993). A negative potential change is related to the initiation of behaviour. A positive potential change is related to performing cognitive tasks or in different situations in cognitive inactive states. It can also reflect inhibitory processes or interruption of ongoing activity. (Elbert, in McCallum, 1993). Nowadays the term 'SCP' often appears. Terms as 'the Bereitschaftpotential' and Contingent Negative Variation (CNV) are also often used in literature. Both are slow cortical potentials, but the terms are linked to the specific experimental design in which they appear.

In general, the functional significance of SCPs is that they are a measure of cortical excitability. Negative SCPs reflect an increase in excitability of the underlying cortex, whereas positive SCPs reflect a decrease in cortical excitability. Negative SCPs and paroxysmal depolarization shifts in cortical neurons occur simultaneously (Birbaumer et al, 1990). This confirms the hypothesis that a negative SCP indicates an increase in cortical excitability. Further research on the epilepsy model shows that negative SCPs reflect widespread depolarization of apical dendrites and these decrease thresholds of paroxysmal activity. This implies that seizure probability increases when there is more paroxysmal

depolarization (Birbaumer et al, 1990), as was also mentioned above.. Further evidence for negative SCPs and lowered excitability thresholds is the finding that epileptics can produce a negative potential shift by means of hyperventilation which is a common technique used to induce a seizure. Additionally, it turned out that this induced negative SCP is reduced by anti-epileptic medication. (Rockstroh et al., 1993)

Epileptics have a disturbance in the regulation of cortical excitability (Rockstroh et al.,1993). Self-regulation of SCPs in epileptics has advantageous effects on seizure control. Many epileptics can feel seizures coming just before the actual occurrence of the seizure for example by visual or auditory auras. This awareness before a seizure is accompanied by a negative SCP. This negativity can be regarded as a sign of cortical over-excitability. When this happens, regulation towards positivity can counter upcoming epileptic negativity. It thus lowers the growing excitability, and can therefore prevent a seizure.

SCPs were described in scientific literature from the beginning of the 1960's (Walter,1964). Due to inadequate electronics and a lack of good filter systems these first SCPs were not reliably measured and these studies were severely criticized. There has been great development in DC EEG systems and from the mid 1980's it was possible to reliably measure SCPs. Good studies are reported from that period. SCPs are prone to artifacts like eye-movements, temperature variation and sweat gland potentials (see appendix 1). With regard to SCPs and epilepsy there are two groups of researchers who pioneered in this field.

In a study performed by Rockstroh et al. (1993) twenty-five patients with drug-refractory epilepsy and different types of seizures participated. They received 28 neurofeedback sessions in which they learned by operant conditioning to produce a negative SCP or to suppress these, depending on the condition. Each session consisted of 110 trials with a duration of 8 seconds. Epileptics performed worse on SCP regulation than healthy participants. This is another indication that epileptics have a disturbance in the regulatory skill of cortical excitability. Seventeen out of twenty-five participants could differentiate between the two conditions. Eighteen eventually kept record of the number and severity of their seizures in a diary. After one year 6 participants turned out to be seizure-free, 7 participants had a significant reduction in seizures and 5 participants had no significant changes in their seizures. These groups did not differ in their ability to regulate their SCPs. However, patients who were seizure free after one year performed 5 to 20 times better in the transfer condition. This result indicates that the

degree of seizure reduction is dependent on the performance in transfer trials. Furthermore, there is a tendency for young patients to perform better and it seems that the degree of learning is motivation dependent. From this study it is unclear to what extent type of epilepsy contributes to the seizure-reduction. However, from a presentation by Hinterberger (personal communication, 2005) some evidence arose that simple partial seizures seemed to respond better to SCP training rather than complex seizures.

Kotchoubey et al. studied the effects of SCP neurofeedback in a group of epileptics (Kotchoubey et al., 1996). Twelve drug-refractory patients participated in this. Five of them suffered from complex focal seizures, five from simple focal seizures and two from generalized tonic-clonic seizures. In six patients the locus was on the right hemisphere. For the other six there was uncertainty about the location. Patients turned out to be able to generate positive and negative SCPs and to differentiate between them. There was a significant effect of neurofeedback on the strength, duration and number of seizures. The conclusion is that generating positivity is responsible for this improvement of their epilepsy.

About one third of the patients did not have any reduction in seizures frequency. In a replication study, in which 27 focal epilepsy patients participated, the patients showed larger amplitude differences in their SCPs before onset of the training. These amplitude differences turned out to be the best predictor for beneficiary effects of SCP neurofeedback (Kotchoubey, 1999). This suggests that it can be predicted beforehand which patients will be successful in SCP neurofeedback. Out of 27 patients, nine patients had a right focal focus, seven had a left focal locus and eleven had a multi-focal locus. In the study, no remarks were made about the relation between focus of epilepsy and effectiveness of SCP neurofeedback sessions.

In the above mentioned studies factors predictive of successful or unsuccessful differentiation of SCP neurofeedback were limited. In those studies, no statistical analyses were mentioned which could provide strong evidence for predictive factors. The effectiveness of SCP neurofeedback has been proven extensively, but it was necessary to better address relevant factors that contribute to seizure reduction. These include seizure type, personality, psychosocial adjustment, SCP characteristics and response on SCP neurofeedback training. Strehl et al. (2005) contributed to this search. Their 44 patients were characterized by focal seizures, intractable epilepsy, a seizure frequency of at least two seizures a month, a minimum IQ of 80 and a minimum age of 15 years. Fifteen of these had a left temporal locus, nine had a

right temporal locus and of ten of them there was uncertainty about the locus. Three factors turned out to be the biggest predictors for successful differentiation: total SCP amplitude before the beginning of the training, the focus of the epilepsy and personality variables. The patients who will have the most benefit from SCP training do not have large negative SCP amplitudes before the SCP training, they score low on life-satisfaction and are reactive to stress. Patients with left temporal lobe seizures are in disadvantage. The group with the complex partial and secondarily generalized seizures benefit most, if they could control their SCPs at the end.

Besides neurofeedback, galvanic skin response biofeedback (GSR) also has been examined as a treatment for epilepsy. GSR is under control of the autonomic nervous system (ANS) and the ANS is under neural control. Therefore, it is assumed that skin conductance variation reflects a mental state change of the brain and it can be influenced by means of GSR biofeedback sessions (Thompson and Thompson, 2003). GSR is a measure of skin conductance which is an index of autonomous sympathetic arousal. A weak electrical current is delivered to the skin and by recording the conduction of that current the GSR is picked up. The conductance rises when sweatglands in the fingers secrete more sweat. There are two characterizations of skin conductance; it can be regarded as tonic or as phasic. Tonic skin conductance reflects the level of conductance when there are no external influences. It only varies to interpersonal variations and autonomic regulations. Phasic skin conductance on the other hand, reflects event-related conductance. Both are indices of voluntary and involuntary changes in arousal which can be associated with emotion, attention and physical activity (Neumann and Blanton, 1970) It is the phasic characterization which biofeedback makes use of. GSR is a DC potential, which means it is measured in the low frequency range ([http://bioweb.usc.edu/courses/2003-spring/documents/bisc230-lab\\_psych\\_physio\\_states](http://bioweb.usc.edu/courses/2003-spring/documents/bisc230-lab_psych_physio_states)).

The effect of GSR biofeedback has been examined in a double-blind study in a group of 18 epileptic patients which was originally funded by BIAL (Nagai, 2004). Ten patients were in an active biofeedback group and 8 patients were in a control group. Of the experimental group, seven had complex focal seizures, two had generalized tonic-clonic seizures and one had generalized absence epilepsy. In the control group five patients had complex focal seizures, two had generalized tonic-clonic seizures and one had idiopathic generalized epilepsy. All patients had seizures despite usage of medication. There was a significant reduction of seizure frequency in the GSR biofeedback subjects compared to controls.

Furthermore a correlation was found between the degree of GSR control and reduction of seizures. Striking in this is that there is no significant correlation between the absolute measure of GSR response and seizure reduction. Acquiring GSR control is critical in achieving seizure reduction irrespective of degree of control. (Nagai, 2004). This study is the first to show that GSR biofeedback can be successfully applied in drug-refractory epileptics to reduce their seizure frequency. GSR biofeedback has an advantage over SCP neurofeedback in that it is easier to apply and implement in software programs. Additionally it is suggested that GSR biofeedback is easier to learn (Nagai, 2004).

Above two biofeedback procedures have been described which both have an anti-epileptic effect. These procedures were examined simultaneously by a different study performed by Nagai (Nagai, 2004). In this study, the relationship between peripheral arousal (as measured by GSR) and cortical arousal (as measured by the amplitude of the CNV) was examined. The difference between peripheral and cortical arousal is, that peripheral arousal is a more behavioral measure. Cortical arousal reflects the central regulation of arousal. Cortical negativity was invoked by means of the Contingent Negative Variation paradigm and was measured in two different physiological states. First, a state of arousal (reflected by a low skin resistance) was invoked by GSR biofeedback. Then, a state of relaxation (reflected by a high skin resistance) was invoked. The amplitude of the CNV as invoked by the CNV paradigm was measured in both physiological states. In the peripheral more relaxed state, higher cortical arousal was measured (larger CNV amplitude) and vice versa. There were 18 drug refractory epilepsy patients in this study. Ten of them got active biofeedback training and eight received sham feedback. These ten in the experimental group turned out to have at least 50% reduction in their seizures. This was a statistically significant reduction as compared to the control group (Nagai, 2004).

These two procedures seem to be equally effective in epilepsy patients, hence one could suspect a common underlying neuronal source. Thus far, one study found in the literature, has reported on the effect of EEG biofeedback training (SCP training) on the QEEG (Kotchouby et al., 1999). They did not find a specific effect of the SCP training on the QEEG. Correlations between spectral EEG parameters and SCP data were low and inconsistent. Kotchouby et al. (1999) concluded that learned SCP shifts did not directly lead to specific dynamic changes of the EEG power spectra. For GSR training no QEEG studies have been done to the author's knowledge.

In the current study, 19 healthy volunteers were assigned to one of two groups; one group received SCP neurofeedback and one group received GSR biofeedback. For all subjects both EEG and GSR were acquired simultaneously, and one of these served as the feedback modality (SCP feedback or GSR feedback) whereas the other modality was passively registered for inspection of the interrelation between the two measures. All subjects underwent pre- and post testing on a QEEG.

Main point of interest is to quantify correlation in time between GSR en SCP and to determine to what extent the GSR signal can be predicted from the SCP signal and vice versa. It is expected from previous studies that peripheral arousal and cortical arousal are inversely related. In order to address the main point of interest; e.g. the interrelationship between the SCP and GSR arousal, we recorded both signals simultaneously. Based on a training session a grand average will be computed, showing the averaged response, where the area under the curve is a measure of learning. The better subjects master the task, the bigger the area between the graph and the X-axis. The grand averages for the passively registered modality (in the case of SCP feedback this is the GSR signal) will also be plotted, and the consequent areas calculated. If there is a functional relation between the SCP and GSR response; these areas should be correlated either positively or negatively. The learning procedure (training) will further strengthen these correlations, since the learning procedure will increase the area over sessions, hence the passively registered modality should then undergo the same modulation. In order to control for individualized learning strategies we calculated these correlations for every individual subject.

Second aim of the study is to test which group performed better on the feedback session. It is hypothesized from the study form Nagai et al.(2004) that GSR biofeedback is easier to control.

Finally, we wanted to investigate whether there were consistent changes to be found in the pre- vs. post-QEEG and whether this would be the same for both treatment modalities, hence suggesting a common underlying neuronal mechanism.

## ***Methods***

### ***Participants***

Twenty healthy subjects participated in a standardized training program. There was one drop-out after the pre-test and the other had to be given new thresholds half way so she did not complete 20 sessions in total and was therefore excluded for analysis. The mean age of the subjects was 23.2 years (range 18 –40 years). There were 8 men and twelve women.

Exclusion criteria were a psychiatric or neurological history, drug abuse or a history of drug usage, use of psychoactive medication. Every participant gave informed consent prior to the study. This study was approved by the regional medical-ethical committee.

### ***Material***

Participants of this study sat behind a desk with a 17” Thin Film Transistor (TFT) monitor in front of them. For the training the software program BioExplorer ([www.cyberevolution.com](http://www.cyberevolution.com)) was used. The EEG was recorded by the Brainquiry PET-EEG and the GSR was recorded by the Brainquiry PET-GSR (also see [www.brainquiry.nl](http://www.brainquiry.nl)). The program was running on a computer where the data was also recorded. As data-analysis software Bioreview, Matlab, Excel and SPSS software were used.

### ***Design and procedure***

In this study there were two groups; one group received feedback on their SCPs and one group received feedback on their GSR signal. From each participant both SCPs (EEG) and GSR were recorded simultaneously independent of group. In each group there were 9 participants, who were equally distributed for gender and age. All completed 20 sessions. The sessions were distributed over 8 weeks and there was at least one day and at maximum 5 days between each session. Before each session drug usage like coffee etc was administrated by a standardized questionnaire (see Appendix 2) and after the session it was administrated how the participant was with the obtained results. (see Appendix 3). Each session had a duration of approximately 45 minutes. It took 15 minutes to attach the EEG and the GSR devices and for the signal to stabilize (see Appendix 1). It took 30 minutes to do the training protocol.

Each session was divided into 4 runs (also see figure 2). After the second run there was a short conversation to reflect on the results of the first two runs and to coach and encourage the participant. Each run consisted of 40 trials of which 24 trials were in the upward direction (U) and 16 trials were in the downward direction (D).

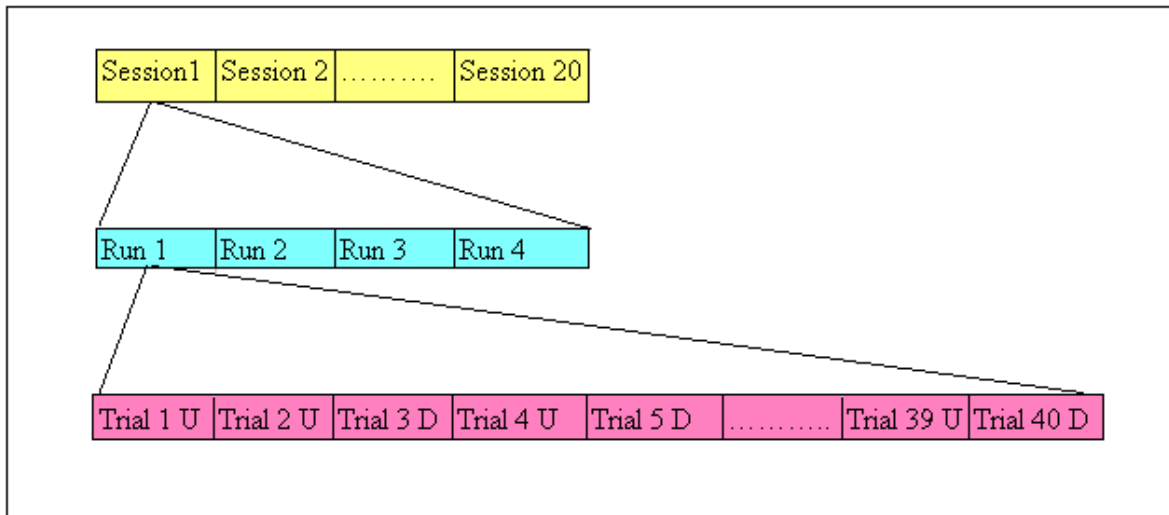


Figure 2: Construction of the training program

Each trial had a duration of 8 seconds and started with a tone (also see figure 3). During the first second the trial type was indicated by the blue square. In the last 500 ms of that first second, the baseline was determined. After that, 7 seconds of feedback took place where the indicating square started white and stepwise filled with blue till the end of the trial. Between two trials there was a random inter-trial interval varying from 1.5 to 3 seconds.

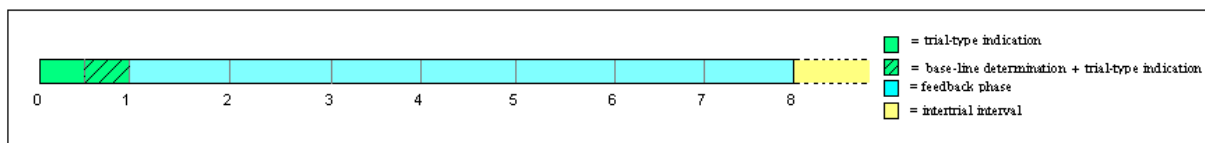


Figure 3: Different phases of a trial

Feedback is defined as seeing the height of your signal represented by the height of a bar (also see figure 4). The feedback for both conditions was similarly displayed on a computer screen in the form of a vertical bar (the yellow bar in fig. 4). The yellow bar is called the feedback bar and the screen in total is called feedback screen. (because it is the screen the participant saw his feedback on). In the training there were two different types of trials. The type of the

trial was indicated by two upper and two lower squares of the screen. Depending which part is coloured blue, indicates the trial type.



*Figure 4: Feedback screen. The blue area indicates the participant should modulate the signal in upward direction. The yellow bar signal reflects the integrated EEG or GSR signal. The numbers show the percentages correct for upward direction (44%) and downward direction (25%). The smileys pop up when the participant exceeds the threshold bar (horizontal lines in middle of the screen) in the indicated area. When the signal exceeds threshold for more than two seconds, the participant hears a reinforcing sound.*

The height of the feedback bar was proportional to the level of the EEG or GSR compared to the 500 ms pre-trial baseline. Dependent on the indicated trial type the participant was instructed to try to modulate either their SCP response in a positive or negative direction or their GSR response in a positive or negative direction (depending on the trial type). Upward movements of the feedback bar indicated an increase in negativity of the SCP signal above the mean of the 500 ms pre-trial baseline, downward movements of the feedback bar indicated a decrease in negativity of the SCP signal. For the GSR condition an upward movement of the feedback bar meant an increase in peripheral arousal, a downward movement meant a decrease in peripheral arousal.

Every participant obtained his own personal settings which were determined from two pre-training sessions. This was done by playback of the first eight runs, while parametrically varying the thresholds. This resulted in average percentages correct at five different threshold settings. The 33% points were determined as personal settings for every participant by linear approximation so that every person could start at his own personal level and had a start rate of 33% correct for both trial types. This way every participant could acquire the same amount of

learning. Besides giving every person his personal setting there were 6 different design versions. They differed from each other by sequence of upward trials and downward trials and sequence of inter trial intervals, so there would be no anticipation effects. These 6 designs were randomised over sessions and counterbalanced across groups.

Prior to and after the training sessions all participants had a qEEG pre-test and a qEEG post-test.

## **Pre- and post QEEG**

The test battery lasts for approximately 60 minutes, and will be undertaken with simultaneous EEG, skin conductance level (SCL), skin conductance response (SCR), heart rate (HR), respiratory rate (RR), muscle tension (EMG) and reaction-time (RT) measures. For this study we only investigated Eyes Open and Eyes Closed EEG data.

## **Neurophysiology Recording protocols**

Subjects are seated in a sound and light attenuated room with an air-conditioned ambient temperature of  $24\pm 1^{\circ}\text{C}$ . An electrode cap is used to acquire data (Nuamps) from the Fp1, Fp2, Fz, F3, F4, F7, F8, Cz, C3, C4, T3, T4, T5, T6, Pz, P3, P4, Oz, O1 and O2 scalp sites. Linked mastoids serve as reference. Horizontal eye movement potentials are recorded using two electrodes, placed 1cm lateral to the outer canthus of each eye. Vertical eye movement potentials are recorded using two electrodes placed on the middle of the supraorbital and infraorbital regions of the left eye. Skin resistance at each site is  $< 5 \text{ k}\Omega$ . A continuous acquisition system is employed and data was EOG corrected offline. Gratton procedure and EOG thresholding for contaminated epochs (exceeding  $\pm 100 \mu\text{V}$ ) are automatically rejected. The sampling rate is 500Hz. A 70 Hz low-pass filter is applied to the signals prior to digitisation.

This method has been published elsewhere in more detail by Gordon, 2003.

## ***Physiological recordings***

For the recording of the GSR and SCP feedback the software program BioExplorer was used. The EEG was recorded from Cz. Linked mastoids were used as reference and a ground was placed on the left side of the forehead. Active Ag/AgCl electrodes were used for EEG recording. Ten20 paste was used as an electrolyte only at Cz. Vertical eye movements were monitored via active Ag/AgCl electrodes that were filled with electrode jelly. These electrodes were placed 1 cm above and below the right eye. All electrode sites were first prepared with Nuprep in order to clean it and to make the location a little rough in order to reduce impedances. The SCPs were computed from the EEG by a moving average of 500 ms that was made every sample of the EEG, which has a rate of 200 samples a second. The GSR was recorded from the third and fourth finger of the non-dominant hand using the BraInquiry PET GSR which has two stainless steel electrodes. For cleaning the middle and ring finger alcohol swabs were used. The PET GSR has a range of 0-10 M $\Omega$  and has a sensitivity of 5-50 k $\Omega$  with a two percentage deviation. The GSR signal was sampled at a rate of 10 per second. The GSR signal was filtered with a high pass first order butterworth filter with a cut-off frequency of 0.5 Hz.

### ***Data reduction and analyses***

There were three sets of data; 1) behavioral data as reflected by the percentages correct reponses, 2) physiological data as reflected by the grand-averages across trials and 3) within subject data.

Invalid trials, in cases when there was too much artifact or where impedance was too high, were not included in the analysis. In case when there were more than 25% invalid trials within a run, the whole run was excluded. In an exceptional case where two runs had to be rejected, the whole session was excluded for analysis.

1) Percentages correct responses per run were averaged across sessions and ‘Upward’ and ‘Downward’ trials were plotted separately. For both groups two one-way ANOVA’s were performed for each trial-type separately. Percentage - dependent on upward or downward trials - served as dependent variable, session as a covariate and number of participant as a random-factor. Missing values due to too many invalids were filled up by linear interpolation.

For each analysis T-statistics, p-values, B-coefficients and Standard Errors were determined. These tests give an indication of the learning effect as measured with behavioural variables.

2) For analyzing SCP and GSR differentiation (a physiological measure of learning) all trials were averaged for a period of 4 sessions, and thus a Grand-average was obtained for the specific trial. By taking averages there is corrected for random fluctuations. A second advantage of these grand averages was that it led to data reduction (for performing analysis of ANOVAs a reduction of degrees of freedom was required). This was done separately for the 'Upward' trials and for the 'Downward' trials. Surface between the 'Upward' and 'Downward' graph was determined as being a physiological measure of learning. After a trial started subjects had 7 seconds to show the required response. The grand-averages were obtained by averaging all trials for 4 sessions. 'Surface' was hence calculated as the area between the grand-average for the up vs. down condition or as the difference between a single condition and the x-axis. This grand average showed the averaged response over time for all those trials. By dividing this surface for condition, analyses for 'Upward' and 'Downward' trials were also computed. The effects were determined by different analyses of variance depending on point of interest. As between-subject factors group (SCP trained vs. GSR trained) were used. As within subject factor 'block' (dependently 5 time-frames) and polarity (Upward vs. Downward) were used.

3) For each participant averages per session and per trial-type (up-trial or down-trial) were computed. Surfaces between averaged up-trials and the zero-line and between averaged down-trials and zero line were used as a measure of learning. This was performed for either the GSR (in the GSR trained group) or the SCP (in the SCP trained group), for either condition (up or down). Consequently; the grand averages were computed for the GSR occurring during SCP learning; and the SCP response during GSR learning. By plotting these two measures against each other we could investigate whether these measures were correlated or not.

For each participant correlation between the learning measure (depending GSR or SCP) and simultaneously recorded averaged surface measure of the other variable was computed for each trial type apart. Pearson's correlations and p-values were computed. Significance-level was set on 0.1.

## ***Results***

### ***Analyses of pre- and post QEEG data***

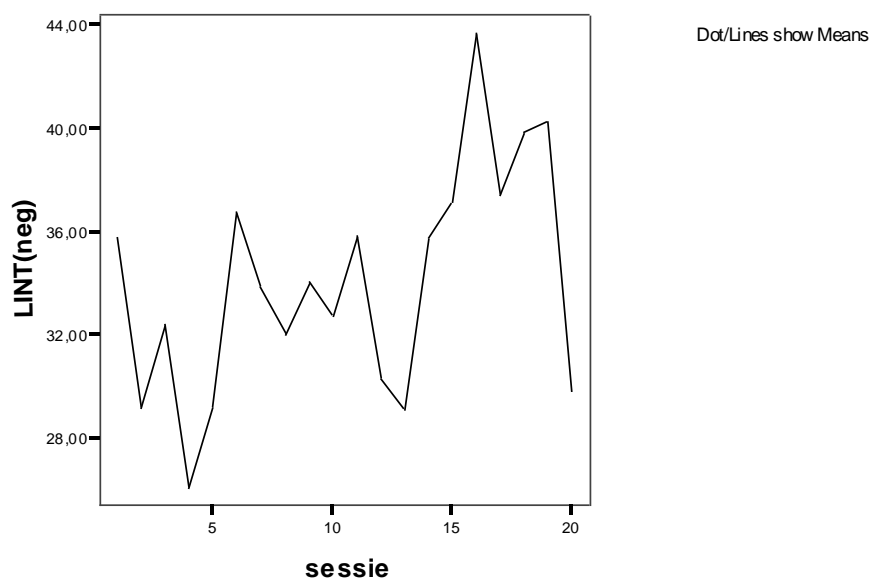
For all participants a QEEG report was obtained for the pre- and post QEEG assessment. Inspection of these reports did not reveal any consistent pattern of change between the pre- and post measurement for both Eyes Open and Eyes Closed condition for all 25 EEG channels and for GSR during these conditions. Since no consistent changes were found, LORETA analysis was not performed, since there was no consistent pattern to localize the source of. Due to the lack of effects in the QEEG we have focused our analysis mostly on the functional intercorrelation between the simultaneously recorded EEG and GSR as will be discussed in the following section, since that method should demonstrate a functional interconnection between both modalities if it exists.

## *Analyses of behavioral data*

For each variable a one-way analysis of variance was performed as discussed in the methods section. Significance-level was set on  $\alpha = .05$

### SCP 'Downward trials' - positivity required trials

There was a non-significant effect of the B-coefficient of session ( $F(1,170) = 0.793, p = 0.374$ )



*Figure 5: The percentages correct for the SCP group for the 'positivity required' trials. There was no significant learning effect for these trials.*

### SCP 'Upward trials' – negativity required trials

There was a significant effect of the B-coefficient of session ( $F(1,170) = 4.597, p = 0.033$ )

	B-coefficient	Std. Error
Intercept	35.445	3.452
Sessie	0.366	0.171

*Table 1: variables of the regression-line*

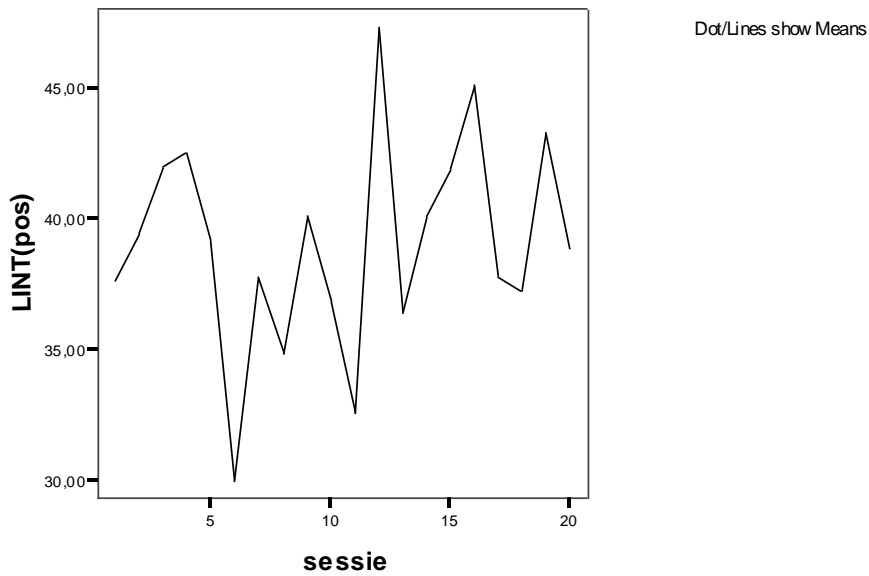


Figure 6: The percentages correct for the SCP group for the ‘negativity required’ trials. There was a significant learning effect for these trials, however, as can be seen from the percentages correct the group as a whole did not learn it up to a very high percentage correct (average 45%).

GSR ‘Downward trials’ – decreased GSR required trials

There was a significant effect of the B-coefficient of session ( $F(1,170) = 3.975, p = 0.048$ ).

	B-coefficient	Std. Error
Intercept	33.034	3.558
Sessie	0.351	0.176

Table 2: variables of the regression-line

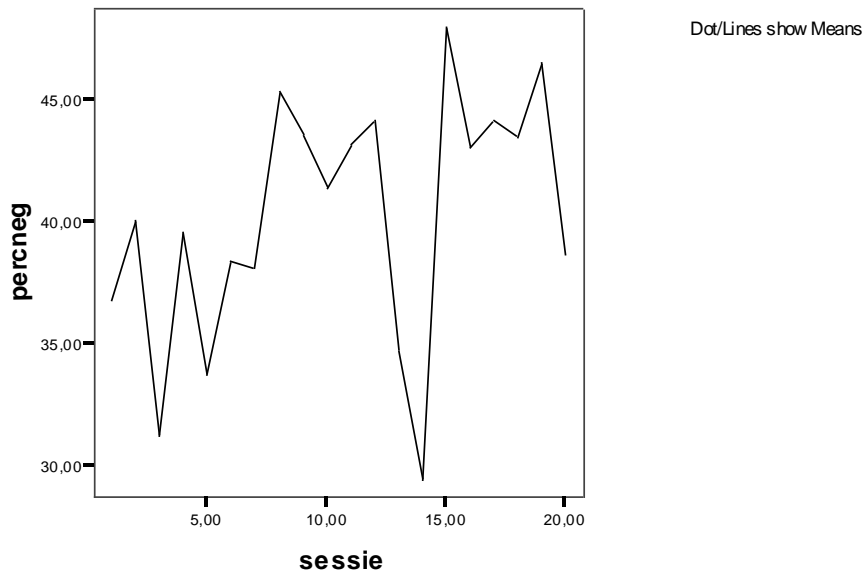


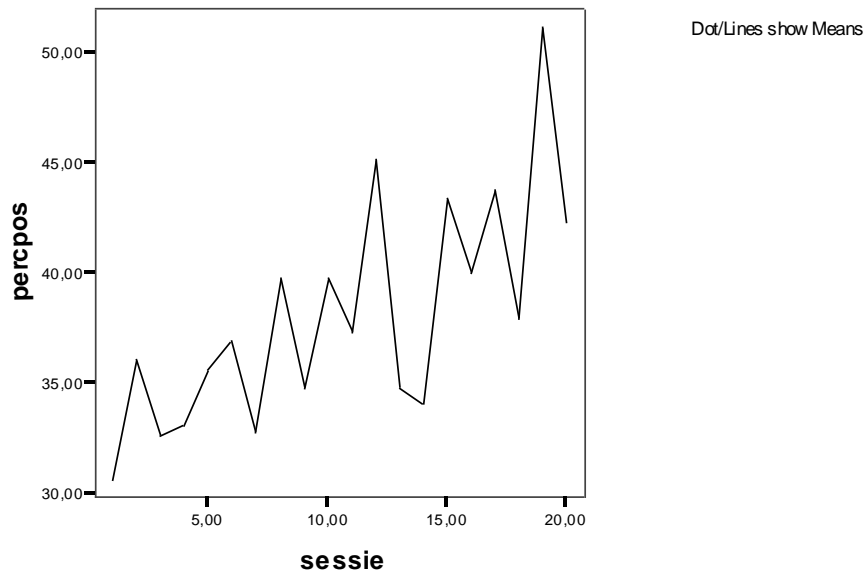
Figure 7: The percentages correct for the GSR group for the ‘ decreased GSR required’ trials. There was a significant learning effect for these trials, however, as can be seen from the percentages correct the group as a whole did not learn it up to a very high percentage correct (average 45-50%).

GSR ‘Upward trials’ – increased GSR required trials

There was a significant effect of the B-coefficient of session ( $F(1,170) = 16.597, p < 0.001$ ).

	B-coefficient	Std. Error
Intercept	45.665	3.035
Sessie	0.611	0.150

Table 3: variables of the regression-line



*Figure 8: The percentages correct for the GSR group for the ‘ increased GSR required’ trials. There was a significant learning effect for these trials, which can also be clearly seen from the figure above.*

In appendix 3 and 4 the individual learning curves for all subjects can be found.

## *Analyses of physiological data*

Three sets of analyses of variance were performed. Significance-level was set on  $\alpha = .05$ .

### *First set of analyses of variance*

Within-subject tests of linear contrasts were used to test for any linear effects of within-subject 'block'. This was tested separately with the SCP data for the SCP-trained group and with the GSR data for the GSR-trained group. As a measure of effect size 'partial eta squared' was used. For the SCP trained group there was a non-significant linear contrast for block ( $F(1,8)=.774$ ,  $p = .405$ ). The effect size was .088 which means 8.8% variance can be explained by the within-subject factor block.

For the GSR trained group there was a near-significant effect for the linear contrast ( $F(1,8) = 4.300$ ,  $p = .072$ ) The effect size was .350 which means that 35% of total variance can be explained by factor 'block'.

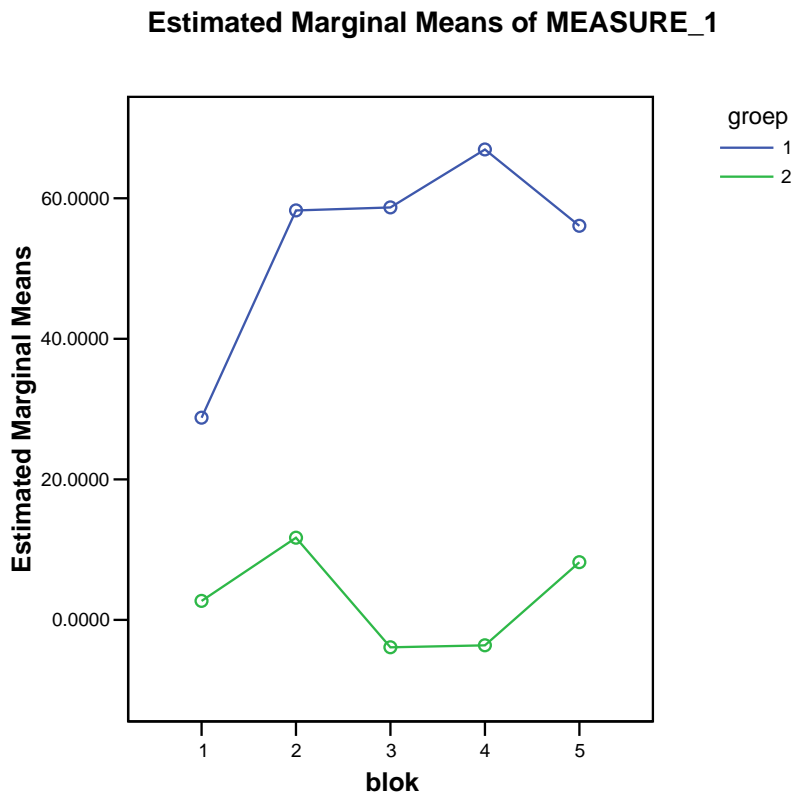
### *Second set of analyses of variance*

Taken all participants together two additional analyses of variance were performed apart for each physiological variable. In these analyses SCP data and GSR data are compared between both groups. 'Block' was taken as within-subject factor and 'Group' was taken as between-subject factor.

There was a significant main-effect of group ( $F(1,16) = 15.746$ ,  $p = .001$ ). SCP-trained participants showed a larger differentiation as compared to GSR-trained participants on the SCP averages. (53.754 vs. 3.020).

Linear within-subjects contrast showed a non-significant effect of 'block' ( $F(1,16)=.560$ ,  $p = .465$ ). Averaged across all participants there is no significant difference over the factor 'block'.

There is a near-significant block\*group-effect ( $F(4,13) = 2.571$ ,  $p = .088$ ). The effect of block is different within the factor group.

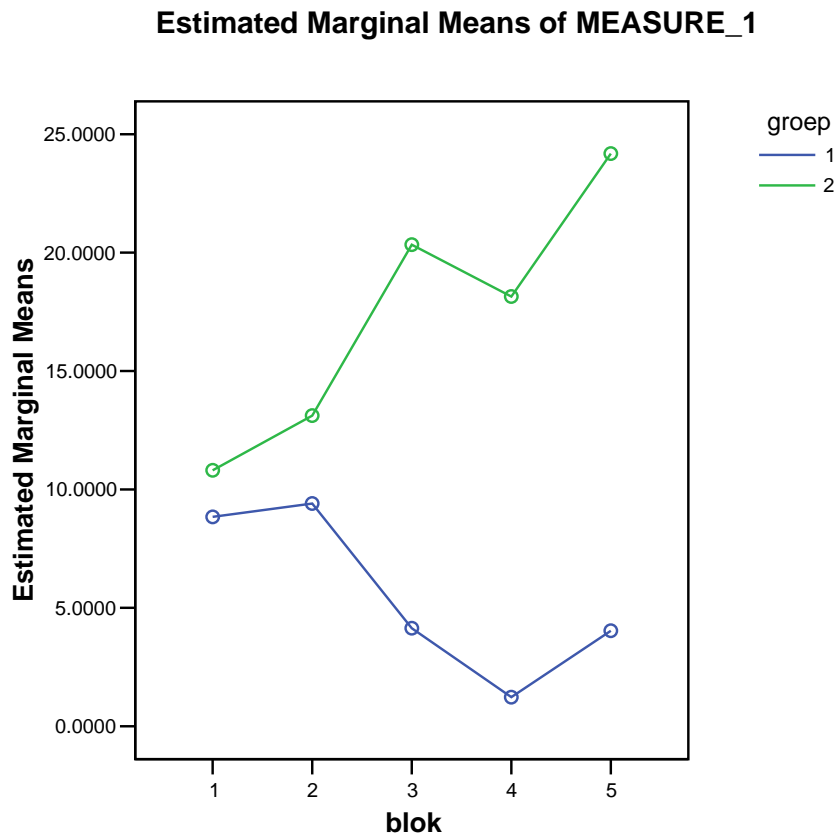


*Figure 9; Differentiation of SCP surface between positivity and negativity trials for the SCP trained group (1) vs. the GSR trained group (2). This figure illustrates that the GSR trained group showed no consistent differentiation over blocks (time). The SCP group showed an increase in differentiation over time, though not significant ( $p=.405$ ; also see ‘first set of analysis’). This illustrates that some learning took place for the SCP learners group with respect to the surface in their grand averages; whereas the GSR learner group showed no consistent increase or decrease in the area of their consequent SCP grand averages, suggesting that SCP learning does affect the SCP grand averages; but GSR learning does not.*

For the analysis of variance restricted to GSR data, again there was a significant difference between groups ( $F(1,16)=6.685$ ,  $p = .020$ ). GSR-trained participants showed a larger GSR differentiation as compared to the SCP-trained group (17.317 vs. 5.530).

Linear within-subjects contrast showed a non-significant effect of ‘block’ ( $F(1,16) = .664$ ,  $p = .427$ ) Averaged across all participants there is no significant difference over the factor ‘block’.

There is a non-significant block\*group interaction effect ( $F(4,13) = 2.358, p = .420$ ). Within group there are no differences between different levels of block.



*Figure 10; Differentiation of GSR surface between increased GSR and decreased GSR trials for the SCP trained group (1) vs. the GSR trained group (2). This figure illustrates that the SCP trained group showed no consistent differentiation over blocks (time). The GSR trained group showed an increase in differentiation over time, which was almost significant ( $p=072$ ; also see 'first set of analysis'). This illustrates that some learning took place for the GSR learners group with respect to the surface in their GSR grand averages.*

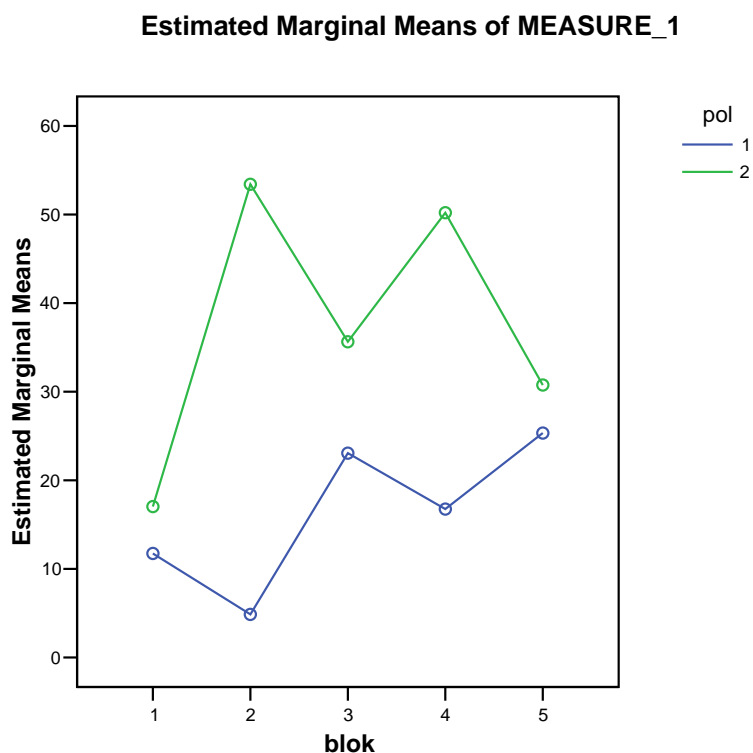
### ***Third set of analyses of variance***

Aim of the third set of analysis was to determine if there is a difference between negativity-required trials and positivity-required trials. Again this was split up for SCP-trained participants and GSR-trained participants. Block served as within-subject factor and polarity served as within-subject factor.

For the SCP-trained participants, there was no significant linear within-subject contrast for 'block' ( $F(1,8)=.774$ ,  $p = .405$ . Effect size is .088 which means 8,8 percentage of total variance can be explained by variable 'block'.

Neither for 'polarity' was a significant effect ( $F(1,8) = 1.439$ ,  $p = .265$ ) SCP-differentiation does not differ between negativity required trials and positivity required trials.

Also no significant block\*polarity effect was found ( $F(4,5) = 1.813$ ,  $p = .264$ ). the effect of 'block' did not differ within different polarity conditions .

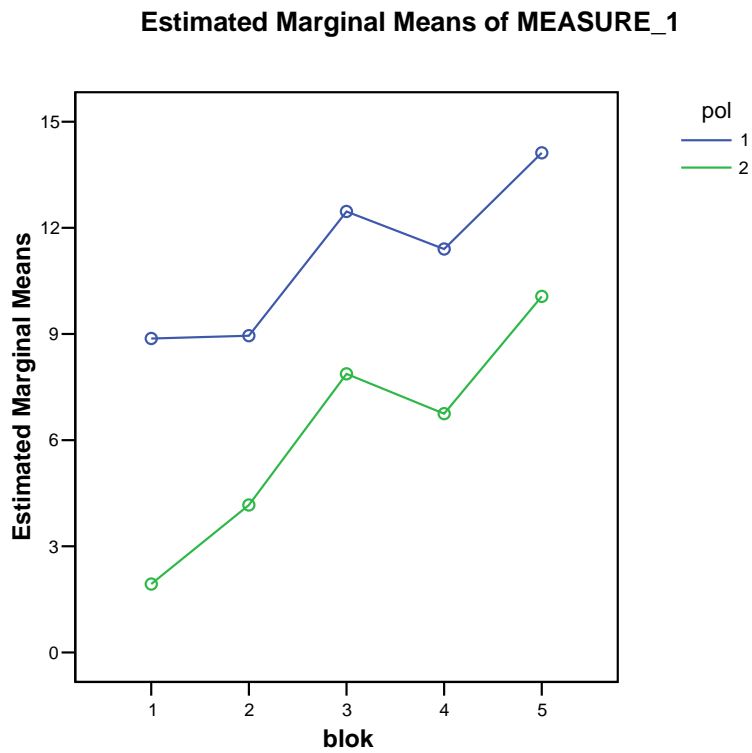


*Figure 4; SCP differentiation for negativity required trials (pol1) vs. positivity required trials (pol2) for the SCP trained subjects. This graph shows that there was a slight increase in surface for both the negativity and positivity trials, though there are no differences between the two conditions, suggesting that no clear learning took place for either condition.*

For the GSR-trained participants there was a near-significant linear within-subject contrast for 'block'. ( $F(1,8) = .4.300$ ,  $p = .072$ ). Averaged across five block there is a linear trend for GSR differentiation. Effect size in this was 0.35.

There was a significant main-effect of polarity; the learning effect of the increased GSR condition differed significantly from the learning effect of the negativity condition. ( $F(1,8) = 14.714, p = .005$ )

There was a non-significant block\*polarity effect ( $F(4,5) = 1.208, p = .411$ ) the effect of 'block' did not differ within different polarity conditions .



*Figure 5; Progress GSR differentiation for increased GSR required trials (pol1) vs. decreased GSR required trials (pol2). This graph shows that subjects learned to increase the surface for both conditions, though better for the increased GSR required trials.*

## Correlations

The graphs below show the individual correlations between the averaged GSR and SCP areas over sessions. Grand-averages per session were made, and the area between the grand-average and the x-axis were calculated. The according grand average of the non-trained modality was computed and the according area was calculated. These two areas were plotted individually over the 20 sessions. If there is a relation between the GSR and SCP it should show up as a significant individual correlation over sessions.

### GSR learners:

Table 4: Correlation between surface averaged GSR Down-trials and the GSR Up-trials with simultaneously recorded averaged surface of the SCP. No significant correlations were obtained between the GSR and consequent SCP for the GSR down trials.

GSR Down trials		
Participant	Correlation	P-value
1	0.155	0.513
2	-.0291	0.214
3	-0.035	0.884
4	0.076	0.772
5	0.125	0.600
6	-0.200	0.397
7	-0.128	0.592
8	-0.286	0.221
9	0.199	0.401
10	0.201	0.395

GSR Up trials		
Participant	Correlation	P-value
1	0.403	0.078
2	-0.020	0.934
3	0.069	0.774
4	-0.315	0.218
5	-0.099	0.679
6	0.117	0.624
7	0.201	0.395
8	-0.094	0.693
9	-0.016	0.948
10	-0.081	0.734

### SCP learners:

Table 5 : Correlation between surface averaged SCP down-trials with simultaneously recorded averages surface of the GSR together with the individual scatterplot for the participant with a significant correlation. The more successful the trial (the bigger the positive area; more positivity); the higher the GSR.

Participant	Correlation	P-value
1	0.341	0.141
2	-0.186	0.432
3	0.384	0.104
4	-0.406	0.076
5	-0.073	0.760
6	0.018	0.940
7	<b>0.485</b>	<b>0.030</b>
8	0.130	0.620
9	0.424	0.063

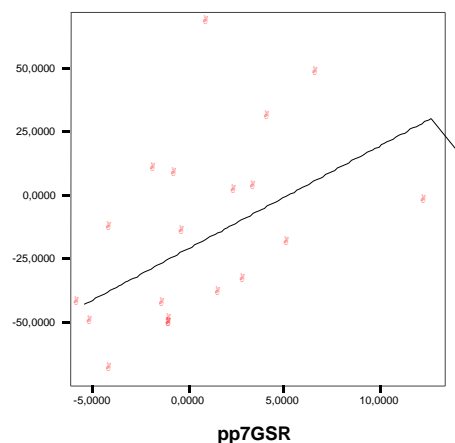
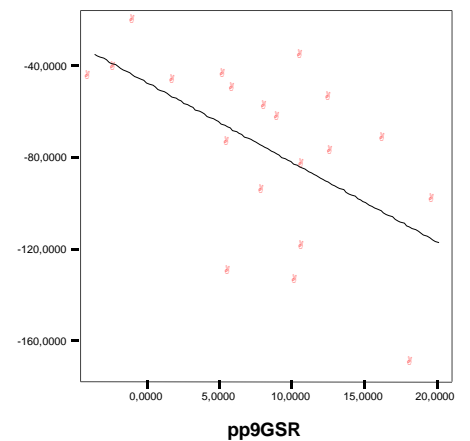
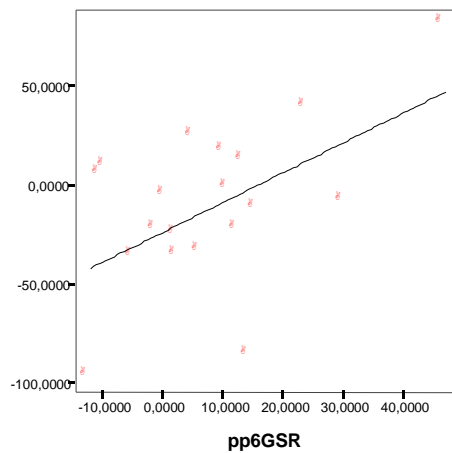
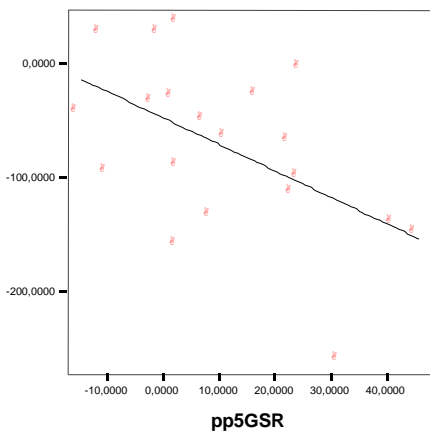


Table 6: Correlation between surface averaged SCP up-trials with simultaneously recorded averaged surface of the GSR. Also see the individual scatterplots for the 3 participants with significant correlations. Two subjects showed a negative correlation. These negative correlations can be interpreted as: The more successful the trial (the bigger the negative area; more negativity); the higher the GSR.

Participants	Correlation	P-value
1	-0.427	0.061
2	-0.017	0.943
3	0.255	0.292
4	0.006	0.980
5	<b>-0.533</b>	<b>0.016</b>
6	<b>0.541</b>	<b>0.017</b>
7	0.049	0.836
8	-0.195	0.453
9	<b>-0.574</b>	<b>0.008</b>



## *Discussion*

In this study two types of biofeedback were examined: SCP Neurofeedback and GSR biofeedback, in which the feedback was presented in an identical way for both modalities.

The learning effects for the SCP group were very moderate as a whole group. From the behavioural data only moderate increases in percentage correct were found up to 44-46% correct whereas every subject started on 30% correct. This effect was only significant for the negativity-required trials. Also, the analysis based on the grand averages using 'area' as a learning measure showed moderate to no effects. The differentiation measure showed that the differentiation between the positivity and negativity required trials was bigger than for the GSR group, though this effect did not increase significantly over time (no learning effect). Furthermore, the differentiation for the negativity vs. the positivity required trials showed no significant effects.

In previous studies using SCP training better learning effects have been obtained than demonstrated in this study. However, it is reported repeatedly that the results of healthy participants on SCP training are disappointing. Kübler (in Hinterberger, 2004) argued that healthy participants may have low subjective attributions of self efficacy. In general it can be concluded that learning of skills without any external or internal perceptual feedback such as brain activity or some autonomic changes such as skin conductance or blood pressure is more difficult as compared with somatomuscular or easy to perceive bodily responses such as heart rate. (Brener in Hinterberger, 2004).

Also the duration of our study (8 weeks) was probably too extended. This may have had a negative contribution. It is known on the basis of oral reports from participants that their motivation seemed to decrease during the progress of the training. Neurofeedback is motivationally dependent (Hinterberger, 1999) and by a lack of motivation participants' self-regulation of SCPs tends to decrease. The fact that subjects seemed to better master the negativity trials as compared to the positivity trials is in line with previous studies. According to Hinterberger (personal communication) subjects and patients learn negativity trials more easily as compared to positivity trials.

It is clear that healthy participants do not have a higher gain by biofeedback sessions, this in contrary to epileptics who possibly can reduce their symptoms. For future studies it could be beneficial to shorten future feedback studies and to add a secondary gain for healthy volunteers like for example an extra bonus by a higher improvement of their percentages correct.

For the GSR group the results looked better. The behavioural data showed better learning curves, which were significant as well, especially for the 'increased GSR required' condition, although the percentage correct still showed moderate effects (approx. 50% correct max.). The grand averages also tended to show increased differentiation over trials, and there were significant learning effects for both the increased and decreased GSR required conditions, though this effect was more significant for the 'increased GSR required' condition. In summary, the GSR group demonstrated significant learning effects and it was easier for subjects to learn to increase their GSR (= increased arousal), though the learning effects were not very strong as measured behaviourally.

For the GSR biofeedback group the trial duration in the GSR designs might have been too short for a GSR to fully develop. The GSR response is a very slow process. If a subject is startled the GSR will increase over a 1-2 second time; and then slowly decrease again. Further inspection of data also showed that at the end of the trial the signal had not reached its maximum value. The 7 seconds used in this study might therefore have been too short to easily demonstrate control over GSR regulation behaviourally. However, the physiological data clearly demonstrated learning effects, though not as strong as expected. The observed effects were most evident for the increased arousal condition. This makes sense in a way that it is easier to temporarily increase one's arousal (such as a startle) rather than decrease one's arousal.

In summary, the results show that GSR biofeedback, as compared to SCP Neurofeedback, is easier to learn which confirms the findings from Nagai et al. (2004). Furthermore, SCP-trained subjects show the biggest SCP differentiation as compared to GSR-trained people and GSR-trained people show the biggest GSR differentiation as compared to the SCP-trained, which confirms that the primary learning effect can be found in the modality being trained; e.g. if subjects are trained on their SCP, then the differentiation of the SCP is also biggest and increases with training time. No clear differentiation effects were found for the 'other'

modality suggesting that training on GSR does not directly affect or modulate SCP's and vice versa as measured with this differentiation method.

No consistent differences were found in the pre- and post QEEG's for both groups. This is in agreement with the study from Kotchouby et al. (1999) who also failed to report consistent changes in the QEEG after SCP Neurofeedback. Jay Gunkelman – a QEEG expert - also expected no changes in the QEEG after application of these two procedures (personal communication). Based on this finding it is hard to draw firm conclusion about the fact whether both methods tap into a similar underlying neuronal circuitry. EEG only records superficial activity of the cerebral cortex, and does not reflect activity from deeper structures such as the Hippocampus, Amygdala etc., hence it could be possible that this effect is mediated on a sub-cortical level. Finally, since people learned self-regulation of these measures it could also be expected that the changes do not appear during passive conditions such as Eyes Open and Eyes Closed condition, but only when the skills are used. In order to investigate the functional relationship between the GSR and SCP further we analyzed in more detail the session data from all participants of the simultaneously recorded EEG and GSR, since during those sessions the acquired skill was used.

The individual correlation analysis revealed no correlations for the GSR Biofeedback group. However, for the SCP feedback group significant correlations were found for both the positivity and negativity conditions. The significant correlations seem to suggest that successful SCP trials are associated with increased arousal. This could well be interpreted as a non-specific effect, since if people are performing good at a certain task, their arousal will increase.

These correlations are probably not related to a functional relation between SCP regulation and GSR regulation – which was the hypothesis to be investigated in this study. In such a case SCP positivity would be related to increased arousal, whereas SCP negativity would be related to decreased arousal or vice versa. Our results suggest increased GSR arousal being related to both training conditions, suggesting a non-specific arousal effect related to degree of success.

In summary, in this study we demonstrated healthy participants were able to learn self-regulation of their SCP's to a moderate degree and self-regulation of GSR to a medium

degree. We could not demonstrate clear functional interrelations between SCP and GSR regulation for both the pre- and post QEEG and the individual session data. This is probably due to the short duration of trials (7 seconds); which made it difficult for participants to demonstrate optimum voluntary control over their GSR. Furthermore, this short duration might also explain the correlations between increased arousal and both SCP conditions. If a longer duration of trials was chosen; these correlations might not have appeared since the GSR arousal-response invoked by the previous successful trial could then have reached baseline again.

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- Wanneer heb je voor het laatst alcohol gebruikt?
  - A) Niet, niet in de laatste 24 uur
  - B) 3-6 uur geleden
  - C) 6-9 uur geleden
  - D) 9-12 uur geleden
  - E) 12-15 uur geleden
  - F) meer dan 15 uur geleden
  
- Wanneer heb je voor het laatst een sigaret gehad?
  - A) Niet, niet in de laatste 24 uur
  - B) Binnen het laatste uur
  - C) 1-2 uur geleden
  - D) 2-3 uur geleden
  - E) 3-4 uur geleden
  - F) 4-5 uur geleden
  - G) 5-6 uur geleden
  - H) 6-7 uur geleden
  - I) 7-8 uur geleden
  - J) meer dan 8 uur geleden
  
- Wanneer heb je voor het laatst recreatieve drugs gebruikt?
  - A) Niet, niet in de laatste 24 uur
  - B) Binnen laatste 3 uur
  - C) 3-6 uur geleden
  - D) 6-9 uur geleden
  - E) 9-12 uur geleden
  - F) 12-15 uur geleden
  - G) meer dan 15 uur geleden
  
- Wanneer heb je voor het laatst gegeten?
  - A) binnen het laatste uur
  - B) 1-2 uur geleden
  - C) 2-3 uur geleden
  - D) 3-4 uur geleden
  - E) 4-5 uur geleden
  - F) 5-6 uur geleden
  - G) 6-7 uur geleden
  - H) 7-8 uur geleden
  - I) meer dan 8 uur geleden

## APPENDIX 2

Naam:  
ID-code:  
Tijd:  
Datum:  
Sessie:

Hieronder staan een aantal vragen.  
Omcirkel het cijfer dat jouw antwoord het beste weergeeft.

- Hoe tevreden ben je over je prestatie?

Heel ontevreden      1      2      3      4      5      6      7      Heel tevreden

- Hoe vermoeiend vond je de sessie?

Helemaal niet      1      2      3      4      5      6      7      Heel erg

- Hoe gemotiveerd was je vandaag?

Helemaal niet      1      2      3      4      5      6      7      Heel erg

- In welke mate heb je het idee dat je je prestaties kunt beïnvloeden?

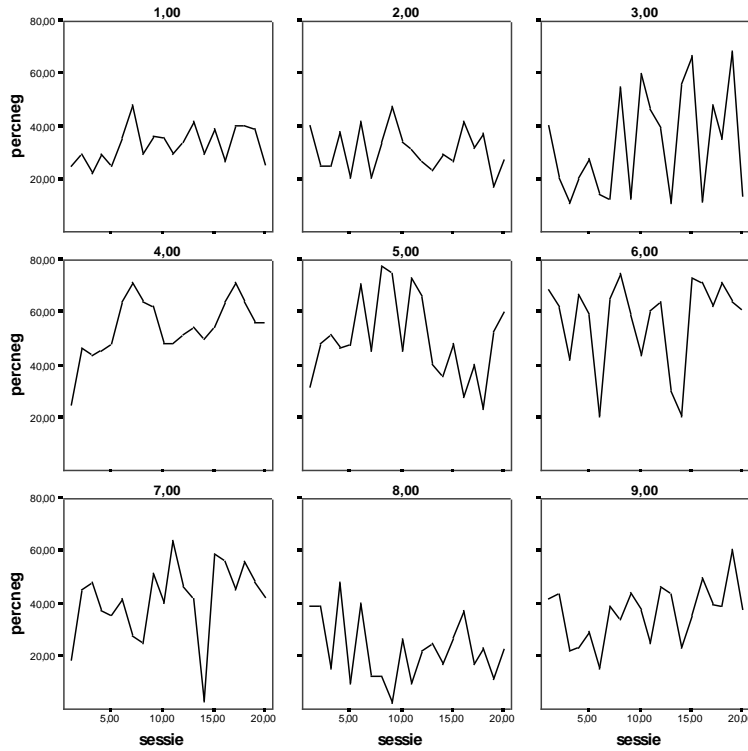
Helemaal niet      1      2      3      4      5      6      7      Heel erg

- Kun je hieronder in een paar zinnen aangeven op welke manier?

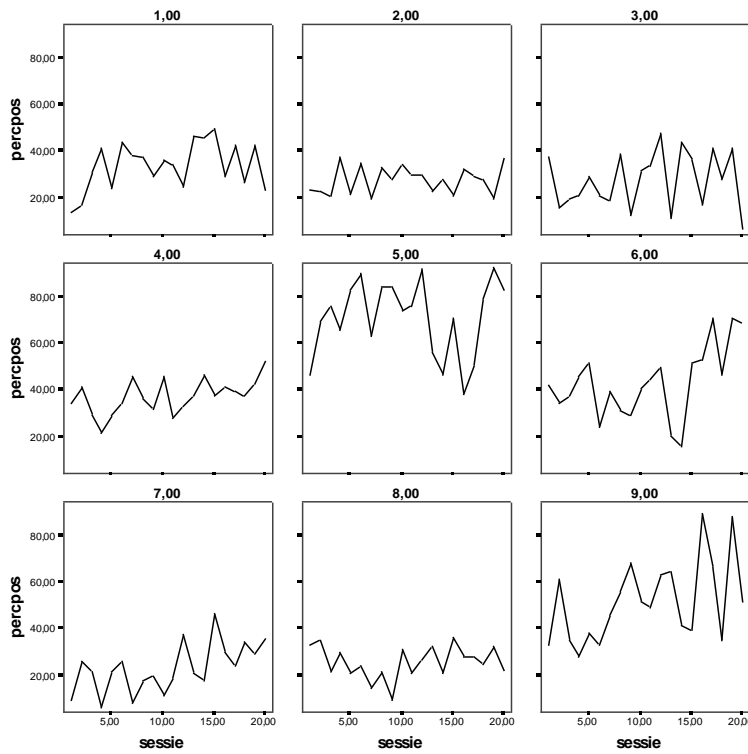
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### APPENDIX 3

*Individual learning curves for the GSR group; top graph is 'decreased arousal' required; bottom graph is 'increased arousal' required.*



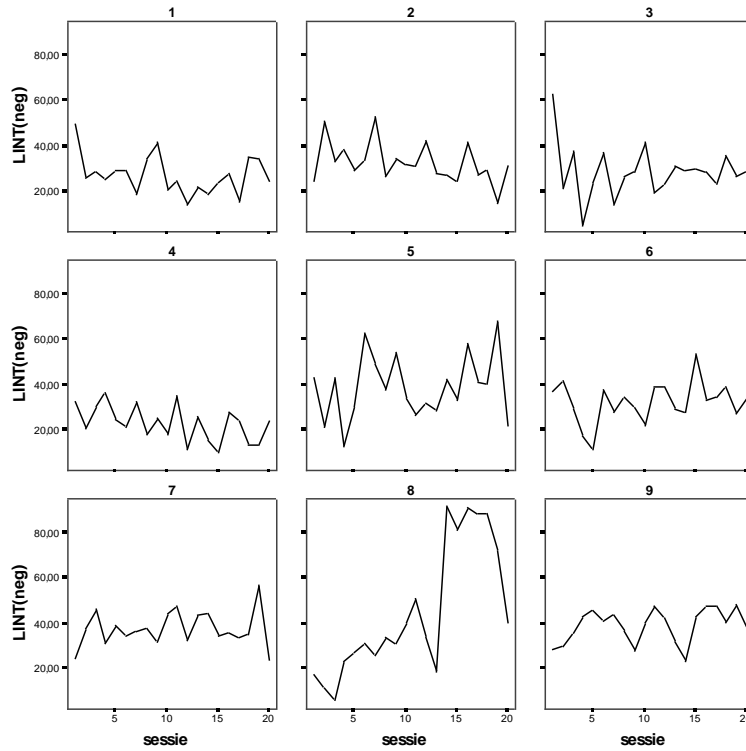
Dot/Lines show Means



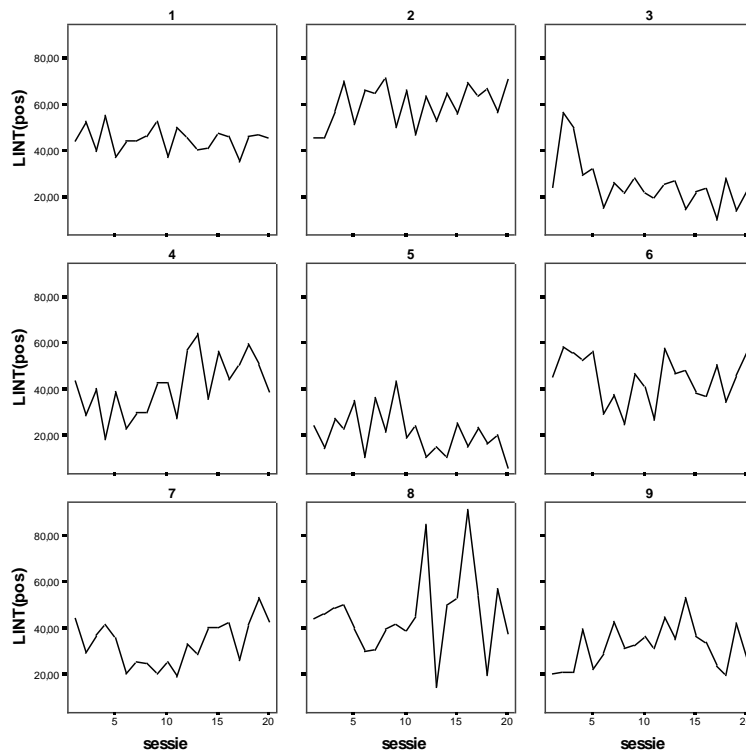
Dot/Lines show Means

## APPENDIX 4

*Individual learning curves for the GSR group; top graph is 'positivity' required; bottom graph is 'negativity' required.*



Dot/Lines show Means



Dot/Lines show Means