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Slow Brain Potentials After Withdrawal of Control

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Summary. The present experiment was designed to replicate and extend the previous finding of an increased postimperative negative slow brain potential shift (PINV) in healthy subjects following an unexpected change from the condition of control over an aversive imperative stimulus to that of loss of control. Two groups of 16 male students each participated in a constant-foreperiod reaction time paradigm with two warning stimuli (WS), each of 6 s duration, followed by two imperative stimuli (IS) of either aversive (loud noise) or neutral (soft tone) quality. The experimental subjects could terminate each IS by pressing a microswitch within 300 ms of IS-onset. After they had experienced this contingency for 40 trials, control was withdrawn in that the IS lasted for 5 s during another 40-trial block, irrespective of the actual motor response of the subject. The yoked control subjects received the same stimuli and performed the same motor response as the experimental subjects, but experienced no contingency between response and IS-termination. EEGs were recorded monopolarly from Fz, Cz, and Pz. In response to the unexpectedly uncontrollable aversive IS, the experimental subjects showed a pronounced PINV over frontal areas, while no comparable PINV developed in yoked controls. Experimental subjects showed no PINV during the first trial block (control conditions), and in response to the neutral uncontrollable IS. Statistical analyses of principle components documented that the PINV can be considered an independent endogenous component.

Key words: Slow brain potentials – Uncontrollability – Principle component analysis – CNV

Zusammenfassung. Die vorliegende Studie untersucht langsame kortikale Potentiale in Reaktion auf einen unerwarteten Verlust von Kontrolle über aversive Stimulation. Zwei Gruppen von jeweils 16 männlichen Studenten wurden Zwei-Stimulus-Reaktionszeit-Bedingungen ausgesetzt, unter denen einer von zwei Warnsignalen von jeweils 6 s Dauer entweder einen neutralen imperativen Reiz (Ton) oder ein unangenehm lautes Geräusch (aversiver Reiz) ankündigte. Versuchspersonen einer Experimentalgruppe konnten

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den jeweiligen imperativen Stimulus (IS) durch Knopfdruck innerhalb von 300 ms abbrechen. Nach 40 Durchgängen dieser Kontrollkontingenz wurde die Kontrolle unerwartet entzogen, indem in weiteren 40 Durchgängen der jeweilige IS unabhängig von der motorischen Reaktion der Vp 5 s dargeboten wurde. Die Gruppe „yoked"-Kontrollpersonen erfuhr die gleiche Reizabfolge und -dauer wie die zugeordneten Experimentalpersonen, jedoch ohne Kontrollkontingenz. Das EEG wurde monopolar vom Frontalkortex (Fz), Vertex (Cz) und Parietalkortex (Pz) abgeleitet. In Reaktion auf den unerwartet unkontrollierbaren aversiven IS zeigte sich bei den Experimentalpersonen eine ausgeprägte frontale postimperative Negativierung (PINV). Eine vergleichbare Negativierung war weder unter Kontrollbedingungen (erster Block von 40 Durchgängen), noch bei Kontrollpersonen, noch in Reaktion auf den unkontrollierbaren neutralen IS zu beobachten. Die Analyse von Komponenten, die durch eine Hauptkomponentenanalyse (PCA) gewonnen wurden, weist darauf hin, daß die PINV als eigenständige endogene Komponente betrachtet werden kann.

Schlüsselwörter: Langsame Gehirnpotentiale - Unkontrollierbarkeit - Komponentenanalyse - CNV

Introduction

EEG recordings during a subjects' performance of a signalled reaction-time task have revealed a slow negative potential shift during the interval between the warning and the imperative stimulus. This phenomenon, designated as CNV (contingent negative variation; Walter 1964) has been extensively investigated during the past two decades, and further analysed for early and late negative components with interstimulus intervals exceeding 4 s (Weerts and Lang 1973; Loveless and Sanford 1974; Rohrbaugh et al. 1976; Simons et al. 1979; for review see Rockstroh et al. 1982). After the anticipated imperative stimulus and/or the instrumental response, the negative potential has generally been found to decrease to baseline or positivity. However, there have also been reports of prolonged negativity following the imperative stimulus in patients suffering from severe behavioral disorders, especially schizophrenia (Dongier et al. 1976, 1977; Timsit-Berthier et al. 1976). These findings have led to further research on the functional significance of this postimperative negative variation (PINV) in psychiatric patients, as well as in healthy subjects (Giedke and Bolz 1980; Lutzenberger et al. 1981; Rockstroh et al. 1979). While the PINV in psychiatric populations was first considered a sign of cerebral dysfunction, the findings of PINVs in healthy subjects under particular experimental conditions suggested psychophysiological processes to underly the development of a PINV. Delaunoy et al. (1978) observed a PINV in healthy subjects, who were first tested under the condition of their motor response terminating the imperative stimulus, and then experienced changes in this contingency, so that the motor responses terminated the imperative stimulus in only two-thirds of the trials. These investigators interpreted the PINV as a signal of the subjects' inability "to resolve the uncertainty of the situation" (p. 356). In our research we detected the development of a PINV after the

occurrence of an unexpected loss of the subjects control over an aversive stimulus (Rockstroh et al. 1979): Experimental subjects first learned to terminate auditory imperative stimuli (IS) of neutral (soft tone) or aversive (loud, raucous noise) quality by pressing a button within 300 ms. Response latencies exceeding 300 ms produced a 5 s presentation of the IS. After a block of 40 trials, this contingency of response and IS-termination was changed, so that each IS lasted for 5 s irrespective of the subject's actual motor response. The group of experimental subjects, who unexpectedly experienced the change from motor control over the IS-duration to loss of this control, was compared to a group of yoked control subjects, who received the same stimuli as the matched experimental partners, but did not experience any control over IS-termination. (Yoked control subjects were just instructed to press the button in response to IS-onset.) Within this paradigm, a pronounced precentral PINV develops in experimental subjects, when the aversive IS is no longer contingent upon the motor response. PINVs are not found during control over the IS-termination, and PINVs do not develop in yoked control subjects. These results suggest contingency processing or contingency reappraisal as the cognitive processes underlying the development of prolonged, postimperative negative SPs. This hypothesis is supported by subjective determinants of the PINV development: 25% of the experimental subjects had not believed in the contingency between fast motor response and IS-termination because of inadequate responding; the physiological responses of these subjects resembled those of yoked control subjects, i.e., they did not show a pronounced PINV. On the other hand, those yoked control subjects who erroneously believed in their control over the IS, showed the PINV. Probably, the contingency between response and stimulus conditions (termination) is learned and stored during the first block of trials. If stimulus conditions suddenly change, the contingency is processed or reappraised, giving rise to prolonged or new negative SPs. The question remains, whether the PINV indicates *contingency* reappraisal, processes that are predominantly attributed to the frontal cortex, or *response adequacy* reappraisal, which would be indicated by a precentral PINV, generated mainly in motor association areas. We previously investigated 13 patients with bilateral lesions of the frontal cortex with the design described above (Elbert et al. 1981). These subjects show a large fronto-central PINV during the first block of trials, when they actually experience a contingency between their response latencies and IS-termination. This PINV decreases across trials, in contrast to the increasing PINV across the second trial block in healthy subjects. These findings suggest an impaired ability of frontal lobe lesioned patients to evaluate the response-outcome-contingency (motor response, IS-termination). Consequently, they are less sensitive to the contingency change during the second trial block, and the PINV decreases with repetition of the stimulus configuration in patients.

We can establish the hypothesis that a frontal PINV indicates processes of cognitive contingency appraisal, as compared to a precentral PINV being an expression for the appraisal of sensorimotor programs in uncertain or ambivalent situations. The present study was designed to clarify these relationships. To evaluate the topographical distribution of the PINV, the EEG was recorded from frontal, precentral, and parietal leads. In contrast to the previous studies, subjects were instructed more precisely about the response-stimulus contingency, to

avoid false beliefs at the beginning of the experiment. Furthermore, the statistical analysis of the component scores resulting from a principal component analysis of the SPs will reveal, whether the PINV can be considered an endogenous component (Donchin et al. 1975, 1978), or whether its variance can be attributed to well-known processes, e.g., P300, CNV-resolution.

Methods

Subjects. Male student volunteers were paid for their participation in the experiment which lasted approximately 1½ h. The sample of 32 was restricted to students who reported to be under no current medication and to have had no cardiovascular or central nervous disorder. Subjects were randomly assigned to an experimental group or a yoked control group. After rejection of subjects for excessive eye movements, each group consistet of 14 subjects.

Design and Procedure. According to the signalled reaction time paradigm, subjects heard as warning stimulus (WS) one of two distinguishable sinusoidal tones of 600 or 1200 Hz, 65 dB, for 6 s. Each WS was immediately followed by one of two imperative stimuli (IS), either an aversive noise of 110 dB or a "neutral" tone of 400 Hz, 65 dB. The contingency between the features of WS and IS was counterbalanced across subjects; the WS appeared at random order within subjects. Intertrial intervals with a mean of 20 s and a range of 14 to 26 s followed the termination of each IS. Experimental subjects could terminate the particular IS by pressing a microswitch within 300 ms after IS-onset. If they responded too slowly or if they responded during the WS-interval, the particular IS lasted for 5 s. Subjects of the control group were yoked to the experimental subjects for presentation order, timing, and duration of the stimuli, but no IS termination contingency was provided. All subjects received a total of 80 paired stimulus presentations in two periods of 40 trials each. After the first block of 40 trials, the stimulus conditions were changed for another 40 trials, so that experimental subjects received each IS for 5 s, irrespective of their motor response.

During the experiment, the subject sat in a comfortable reclining chair in an electrically shielded and sound-insulated room. He held the microswitch in his preferred hand. After electrode application, subjects read the instructions to press the microswitch as quickly as possible at the onset of the IS. The experimental subjects were also given the information that adequately fast responses would terminate the IS immediately. No comparable information was given for control subjects. All subjects were also instructed to fixate their eyes and to avoid eye blinks. Eight practice trials made the subjects familiar with the stimulus sequence and provided the opportunity to practice the motor response. Also, in order to demonstrate the IS termination contingency, all subjects were asked to respond too early in one, and too late in another practice trial. For experimental subjects the IS lasted for 5 s in these trials, while the IS stopped in these trials for yoked control subjects. Subjects were not informed that contingencies changed during the experimental session.

Apparatus and Physiological Recordings. Timing, initiation, and termination of all stimuli were controlled by a PDP 8/e computer. The time between IS-onset and the closure of the microswitch was measured to the nearest 1 ms by the PDP 8/e clock. The physiological responses were amplified by a Beckman type R dynograph, digitized and stored on magnetic type by the PDP 8/e.

The *EEG* was recorded monopolarly from vertex (Cz), frontal (Fz), and parietal (Pz) leads (10–20 system), with a time constant of 30 s (high frequency cut off 30 Hz, 6 dB). Reference electrodes were attached by clips to both earlobes, with each ear shunted with a 10 kΩ resistor and the two resistors connected in parallel to the EEG amplifier. Grass silver disc electrodes, chlorided before each experimental session were used, with Grass paste EC 2 as conducting agent. The recording sites on the scalp were prepared by cleaning with alcohol and removing the outer layers of the skin by gently scraping with a sterile scalpel to achieve an electrode impedance level below 5.0 kΩ. Calibration was with 50 μV square wave pulses. EEG activity

was amplified by a Beckman type 9806 A-C coupler with the time constant modified by changing the capacitor in the time constant selector to 110 μF. Data were sampled at a rate of 100 Hz.

Vertical eye movement *(VEMs)* were recorded in exactly the same way as the EEG. Electrodes were affixed about 1 cm above and below the left eye.

Electrodes for the electrocardiogram *(ECG)* were attached to the lower lateral aspects of the rib cage. Raw ECG was converted to R-R intervals by a cardiotachometer coupler which triggered the PDP 8/e. These interpulse interval data were subsequently converted to a rate per s format, each beat being weighted by the proportion of the second it occupied.

Skin conductance responses *(SCRs)* were recorded via Beckman Ag/AgCl electrodes attached to the second phalanges of the index and middle fingers of the non-dominant hand. Johnson & Johnson K-Y jelly served as electrolyte. A Beckman type 9842 skin conductance coupler, set at 0.5 V constant voltage and DC, was used. Data were sampled at a rate of 20 Hz.

Data Reduction and Analysis. Trials were excluded from the analysis if the mean deviation in the EEG channel during the WS-interval and 1.5 s following IS-onset, with reference to a 1.0 s prestimulus baseline, exceeded 20 μV. When more than one-third of trials for a subject in any condition had to be excluded, data for this subject were rejected. This criterion resulted in data analysis of 28 subjects ($n = 14$ in each group), with a mean exclusion rate of 5% of trials. For EEG slow wave and VEMs 50 sample points were collapsed to one point for representation of the magnitude of the response at subsequent half-s intervals for each trial. These data were further reduced by averaging across trials and subjects. For further statistical analysis, components were computed to describe the course of SPs and VEMs during the WS-interval and the IS-interval: (a) an early component during the WS-interval, defined as the difference between a 1 s baseline and maximum negativity during the first 3 s of the WS-interval; (b) a late component, defined as the difference between the least negative point during the 2.5 to 4.0 s WS epoch and the final point (6.0 s) of the WS-interval; (c) a postimperative component, defined as the difference between the means of the 6.0 to 6.5 s and 7.0 to 7.5 s intervals. For clarification of whether the PINV could be considered an independent (endogenous) component of event-related potentials, the SP data were submitted to a principal component analysis (Donchin et al. 1975)[1] and the component scores were tested on experimental effects.

Heart rate responses were scored as the difference between baseline and: (a) the minimum heart rate (HR) (bpm) during the first 3 s WS (D 1), (b) the maximum HR during the same interval (A 1), (c) the minimum HR during the last 3 s WS (D 2), and (d) the maximum HR during the IS-interval (A 2). Scores of SCRs were the difference between baseline and maximum change of skin conductance level during WS-intervals and during IS-intervals.

The results were statistically evaluated by analyses of variance (ANOVAs) with the between-subjects factor GROUPS (experimental group versus control group), and the within-subjects factors TOPOGRAPHY (frontal versus precentral versus parietal recording), STIMULUS TYPE (aversive versus neutral IS), and PERIOD (first versus second block of 40 trials, i.e., control versus no control). Simple effects were confirmed by *t*-tests.

Results

Response Latency

Mean button-press latency, averaged across all subjects and conditions was 224 ms. The difference between mean latencies to the neutral (247 ms) and the

1 Data of the EEG responses, averaged across 0.2 s interval during 6 s WS-interval and 1.4 s IS-interval provided the basis of the variance matrix. Data were averaged separately for subjects, stimulus qualities, and conditions. Thus the matrix $SP^k(t_i)$ describes the course of the EEG under condition k within 37 time intervals t_i. Covariances were computed across 28 subjects with the time points of the EEG as variables. The number of components to be retained was set at the number of specific values equal to or greater than unity. Components were rotated using normalized varimax criterion. An ordinary least squares fit was performed to obtain the component scores (factor scores) as measure of the contribution of the components to the individual SPs

aversive IS (202 ms) was statistically significant ($P < 0.001$) (see Table 1 for F ratios). Response latency increased across the two periods (control – no control) from 220 ms to 228 ms (main effect of PERIODS, see Table 1). The interaction of GROUPS × PERIODS indicated that this overall increase was mainly due to a pronounced increase in response latency by the yoked control group, whose mean latency increased by 17 ms. None of the experimental subjects failed to interrupt the IS more than twice.

Slow Brain Potential

Figure 1 illustrates the mean SP shifts. During the WS-interval a biphasic negative shift becomes evident over fronto-central regions (Fz, Cz) in both groups.

In response to the IS this negativity decreases in most conditions. After the withdrawal of control during the second trial block, however, the graphs show an additional negative shift (PINV) in experimental subjects, with fronto-central dominance.

Figure 2 illustrates the frontal SPs and the components derived from the principal component analysis (PCA).[2]

SPs during the WS-interval are described by two components: while an *early* loading component attains its negative peak at approximately 0.6 s following WS-onset, the *late* component increases throughout the entire WS-interval. These components correspond to conventionally scored and often described components for interstimulus intervals of several seconds. In contrast to the late component, a second peak after IS-onset is found for the early component (Fig. 2). The early component is most pronounced over the frontal recording site, confirmed by a main effect of TOPOGRAPHY (see Table 1 and Fig. 3). As illustrated by Fig. 2 and confirmed by a main effect of GROUPS, the early component is more pronounced in the experimental than the control group. However, the group difference decreases during the second period (interaction GROUPS × PERIODS). The late component shows its maximum amplitude over the precentral cortex (main effect of TOPOGRAPHY, see Table 1 and Fig. 3). An overall decrease of the late component across trials is documented by a main effect of PERIODS. This decrease is more pronounced at the recording site with maximum amplitude (interaction TOPOGRAPHY × PERIODS).

Two additional components were found that had loadings only during the IS-interval, thus being independent from those described for the WS-interval. As

2 The size of components in Fig. 2 corresponds to their contribution according to the least squares fit for the frontal voltage level. The fit, i.e., the weighted sum of components, is compared to the mean SPs in the upper part of Fig. 2. Curves are displayed separately for groups, stimulus qualities, and conditions

3 The long latency may be due to the experimental conditions: subjects learned to expect the termination of the IS after the button press (exp. Ss) or the reaction time of the yoke mate (yoked control) respectively. A P300 is evoked by this event or IS-interruption, i.e., the P300 is delayed by 200 to 250 ms. If the IS continues as in the second period of trials, the P300 complex in each trial depends on the timing of this interval. The described LPW may therefore be nothing else than the average of not completely time-locked P300 waves. The peak latency is therefore approximately 0.6 s after IS-onset. It should be mentioned, however, that the reported analysis is *not* designed for a P300 detection as the EEG is collapsed to 200 ms points

Table 1. Analyses of variance for response speed (RS), components of SPs, early component, late component, late positive wave (LPW), and postimperative negative variation (PINV), and the postimperative component of vertical eye movements (VEM) with the factors GROUPS (experimental vs yoked control group), STIMULUS TYPE (aversive vs neutral IS), PERIODS (control vs no control), and TOPOGRAPHY (frontal vs precentral vs parietal recording). *F* values are reported for components determined by the principal component analysis *(top entry)* and for conventional scores *(bottom entry in brackets)*. Degrees of freedom are 1/26 and 2/52 (for TOPOGRAPHY). All *F* values with $P < 0.1$ are listed

	RS	Early component	Late component	LPW	PINV	VEM PINV
GROUPS		4.5*			(5.1*)	
STIMULUS TYPE	168.4**		(3.5^t)		4.2*	5.4** (10.1**)
PERIODS	4.1**	(7.5**)	5.6*			
TOPOGRAPHY		159.6** (163.8**)	10.6** (16.6**)	13.7**	32.0** (7.9**)	
G × PE	4.9*	5.8*			3.7^t	
G × TO		2.5^t (3.6*)				
PE × TO		5.3** (5.1**)	2.5^t		10.9** (17.7**)	
G × PE × TO		2.5^t (3.8*)			3.8* (9.5**)	
ST × PE × TO				2.8^t		

^{t}: $P < 0.1$; *: $P < 0.05$; **: $P < 0.01$

the ANOVA confirms distinct patterns for the components, it seems justified to consider them as components not only in terms of the PCA but also in terms of underlying processes (see Donchin et al. 1978). A first postimperative component reaches maximum factorial loading at approximately 0.6 to 0.8 s after IS-onset, with negative values from the frontal, and positive values from precentral and parietal locations. It may be attributed to the late positive wave (LPW) complex.[3] The second postimperative component shows negative values with higher scores frontally than precentrally and parietally (for the main effect of TOPOGRAPHY see Table 1). This component is labeled postimperative negative variation, PINV (see Fig. 3 for the scalp distribution of SP components and VEMs).

The development of the frontal PINV (Fig. 1) during the second trial period, in particular in experimental subjects, is documented by the significant interactions PERIODS × TOPOGRAPHY, and GROUPS × PERIODS × TOPOGRAPHY (see Table 1). Also, *t*-tests indicate that the frontal PINV of the experimental group: (a) is more pronounced in response to the aversive *uncontrollable* than in response to the aversive controllable IS ($t = 6.4$, $P < 0.001$), (b) exceeds the PINV in *yoked control subjects* ($t = 3.5$, $P < 0.01$), (c) tends to be larger in response to the uncontrollable *aversive* than in response to the uncontrollable neutral IS ($t = 1.8$, $P < 0.1$).

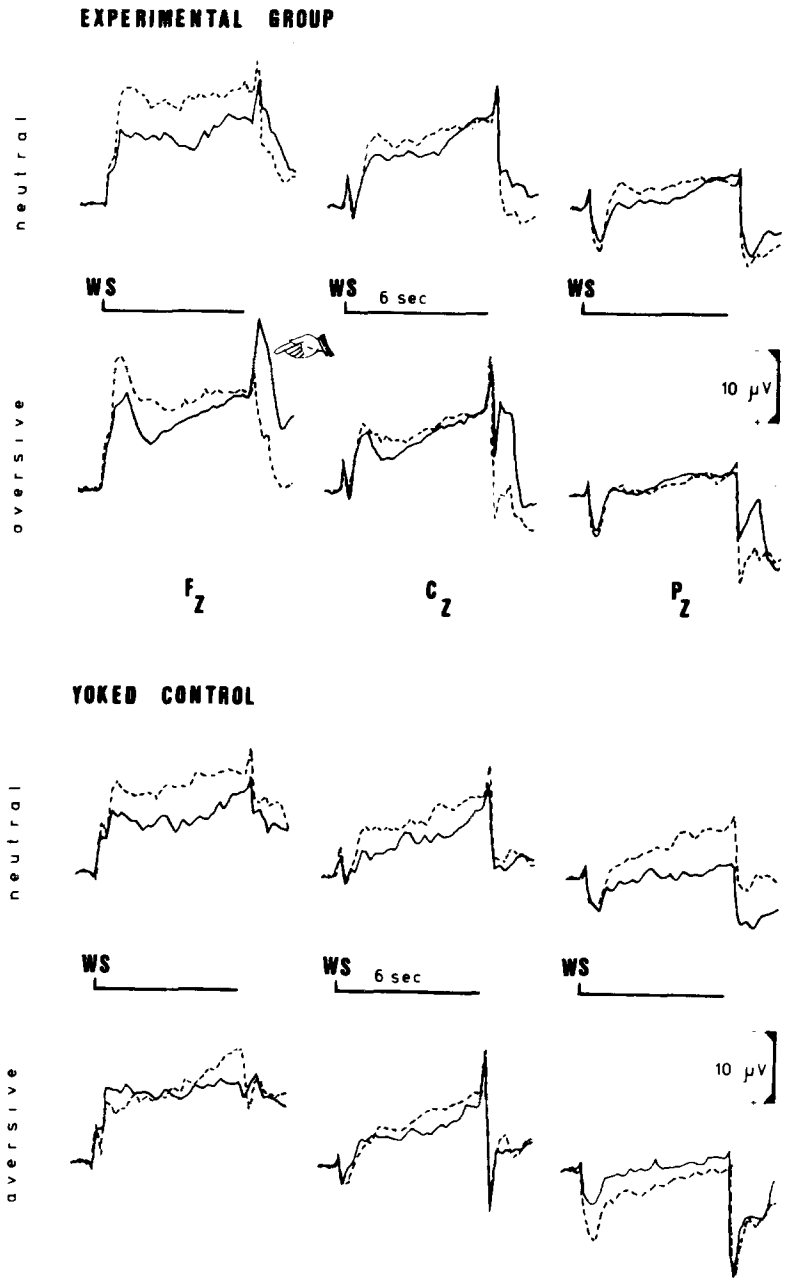
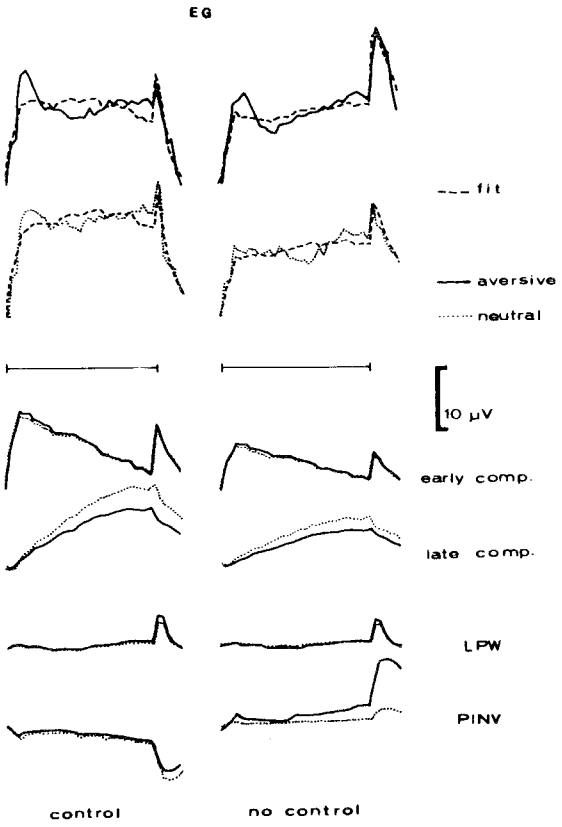


Fig. 1. Slow potentials during the 6 s WS-interval (bar) and 2 s IS-interval, averaged across subjects separately for the experimental group and the yoked control group, for aversive and neutral IS-conditions, the frontal (Fz), precentral (Cz), and parietal (Pz) EEG recording, and for periods (first 40 trials with control in experimental group over the IS by button press within 300 ms (– – – –), second 40 trials after withdrawal of control (———). SP shifts are referred to a 1 s pre-WS-baseline. Note the marked PINV in experimental group after the withdrawal of control over the aversive IS



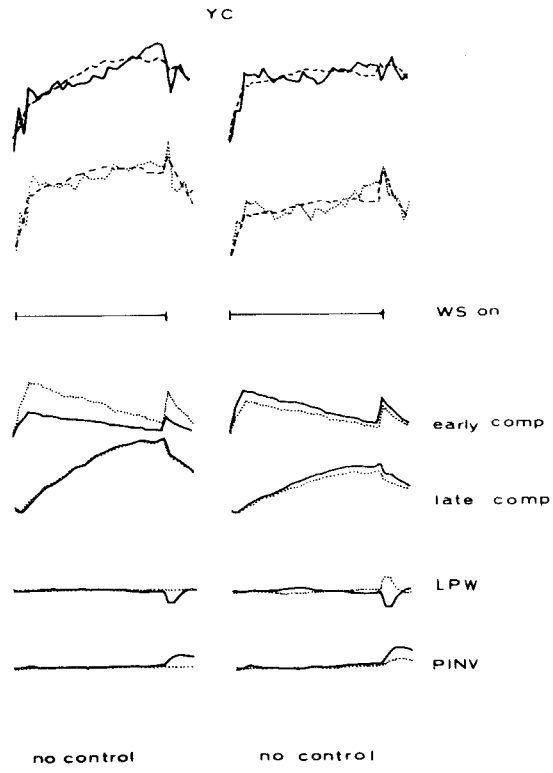


Fig. 2. *Upper part:* Mean frontal SPs during 6 s WS (bar: WS on) and 1.4 s IS-interval for experimental subjects *(left)* and yoked control subjects *(right).* Responses are averaged across subjects separately for aversive *(top entry)* and neutral *(bottom entry)* stimulus qualities and the two periods of 40 trials (control–no control). The weighted sum of principal components is fitted to the SP of every subject and for every experimental condition. The means of these fits are compared to the average SPs *(dashed lines). Lower part:* Components for the WS-interval and IS-interval as determined by the principal component analysis

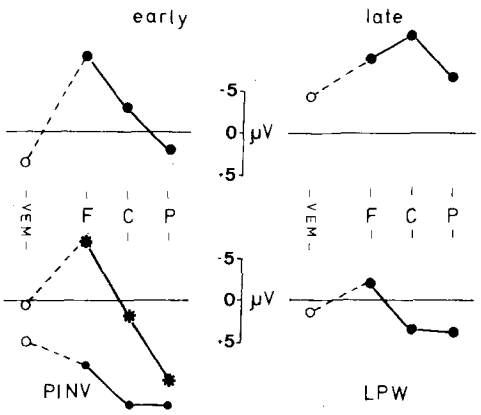


Fig. 3. Topographical distribution of SP components. The mean component scores for the experimental group are displayed separately for the recording sites (F = frontal, C = precentral, P = parietal) and for vertical eye movements *(VEM)*. The VEM scores were determined by fitting the linear combination of SP components to the VEM course. PINV scores are illustrated for the first 40 trials (control ●) and the second 40 trials (withdrawal of control ∗). The μV scale corresponds to the contribution of the component at that point on the time scale at which it shows maximum loadings

Table 1 demonstrates similar results of the ANOVA computed for PCA components and the ANOVA computed for conventional (see p.) components (in brackets). The ANOVA with conventional components provides significant interactions of PERIODS × TOPOGRAPHY, and GROUPS × PERIODS × TOPOGRAPHY, thus supporting the described development of a frontal PINV in experimental subjects under conditions of loss of control. This group difference is, furthermore, confirmed by a main effect of GROUPS.

Vertical Eye Movements

No significant VEM shifts were found with the ANOVA for the WS-interval. A positive shift in VEMs appears somewhat earlier than the first postimperative component, the LPW, and is mainly caused by blink-reflexes. This shift is opposite to the negative going frontal LPW (see Fig. 3), and is more pronounced in response to the aversive IS than in response to the neutral IS (main effect of STIMULUS TYPE: $F(1/26) = 23.5$, $P < 0.01$). Thus, VEMs during the early postimperative interval may counteract the registration of negative shifts, and may have reduced ST effects. The results suggest no crucial influence of VEMs on the reported SP results.

Heart Rate

Mean heart rate (HR) during the WS-interval was higher under aversive than under neutral stimulus conditions (main effect of STIMULUS TYPE). A mean decrease across trials from 73.5 bpm to 71.2 bpm is confirmed by a main effect of PERIODS. Whereas in control subjects this decrease is more pronounced under aversive (3.7 bpm) than under neutral stimulus conditions (2.6 bpm), HR decrease in experimental subjects does not differ between stimulus qualities (decrease of 1.2 bpm under aversive, 1.5 bpm under neutral stimulus conditions). These different tendencies give rise to an interaction of GROUPS × STIMULUS × PERIODS (see Table 2). A triphasic pattern of HR develops during the WS-interval (with D 1 of 1.1 bpm averaged across all subjects and conditions, A 1 of 2.0 bpm, D 2 of 3.1 bpm, and A 2 of 3.1 bpm, referred to baseline). Decelerations

Table 2. Analyses of variance for the components of heart rate – mean heart rate ($\overline{HR}$), first deceleration in the WS-interval (D 1), second deceleration (D 2), first acceleration in WS-interval (A 1), second acceleration in IS-interval (A 2) – and for the components of skin conductance responses during the WS-interval (SCR WS) and during the IS-interval (SCR IS) with the factors GROUPS (experimental vs yoked control group), STIMULUS TYPE (aversive vs neutral IS), and PERIODS (control vs no control). *F* values refer to conventional scores. Degrees of freedom are 1/26. Only *F* values with $P < 0.1$ are listed

	$\overline{HR}$	D 1	D 2	A 1	A 2	SCR WS	SCR IS
GROUPS		6.7*	6.2*				
STIMULUS TYPE	6.0*			4.1*	8.7**	31.1**	52.5*
PERIODS	17.6**	4.5*		11.0**	5.4*	17.0**	
G × PE		5.2*	5.2*	7.1*	8.3**	4.5*	
ST × PE						4.3*	
G × ST × PE	7.7**						

turn out to be more pronounced in control, than in experimental subjects (main effects of GROUPS for D 1 and D 2). The first deceleration increases across periods in both groups, as is documented by a main effect of PERIODS. However, both decelerations increase more in control subjects as compared to experimental subjects (see Table 2: interactions GROUPS × PERIODS).

HR accelerations during the WS-interval and during the IS-interval (A 1 and A 2) are larger under aversive stimulus conditions than under neutral ones (main effects of STIMULUS TYPE). Significant main effects of PERIODS support the reduction of HR accelerations across trials. An interaction of GROUPS × PERIODS points to a more pronounced decrease of A 1 in experimental subjects than in controls. The overall reduction of A 2 is mainly due to a more pronounced decrease in control subjects (from 4.0 to 2.3 bpm), while A 2 slightly increases in experimental subjects (from 3.0 to 3.2 bpm). This interaction suggests that the loss of control over the IS counteracts a decrease of A 2.

Skin Conductance Responses

SCRs during the WS-interval, as well as during the IS-interval are more pronounced for aversive than neutral stimulus conditions (main effects of STIMULUS TYPE). SCRs during the WS-interval significantly decrease across trials (main effect of PERIODS), and this decrease is more pronounced for the larger SCRs under aversive stimuli (STIMULUS × PERIODS).

Discussion

The results of the present study confirm the development of a PINV in subjects experiencing a sudden loss of acquired control over an aversive stimulus. In agreement with previous findings (Rockstroh et al. 1979), no comparable PINV occurred in response to the controllable IS. The postimperative negativity is significantly less pronounced in yoked control subjects, who have not acquired and

expected control over the IS. Furthermore, the present results confirm that the PINV has to be considered an independent endogenous process or component, the variation of which cannot be attributed to the components within the WS-interval. In the present study the most pronounced PINV was recorded from the frontal as compared to the precentral and the parietal sites.

It would be tempting to speculate about a superior frontal contingency "comparator" as the process underlying the PINV. This assumption would be in line with results reported for humans (Luria 1973) and animals (Skinner and Yingling 1977). Loveless (1979), reviewing research on event-related slow potentials, concluded that the "frontal cortex is involved not only with registration of the cue but also with registration of response outcome and therefore that frontal activity might also be found following the response in the RT-paradigm" (p. 87).

However, recorded SPs are not necessarily generated within the frontal lobes. In animal studies, Simson et al. (1977) found that acoustic stimuli generated electrical sources in the insular part of temporal lobes, so that a dipole, oriented perpendicular to the upper temporal plane, would point toward the upper frontal surface, simulating a wave of frontal origin. Little data from humans are available from intracranial recordings. Storm van Leeuwen and Kamp (1973) observed bursts of beta activity in the frontal orbital and cingulate cortex when the subject was uncertain about future events. The activity seemed to be associated with the possibility of outcome evaluation rather than with the possibility of prediction. In the present experiment the precentral PINV was found to be small compared with the previous study. This result might be explained by the modification in subjects' instructions: extensive instruction and practice trials prevented control subjects from believing in control over the IS and assured that experimental subjects realized their response-outcome during the first period. Indeed, postexperimental interviews revealed that all subjects realized the correct contingencies in the present study in contrast to the previous one.

The frontal dominance and the precentral reduction (as compared to the previous study) of PINV amplitude must be attributed to this difference in contingency reappraisal, since other independent variables were kept constant for both studies.

It can be assumed that a PINV appears whenever the response outcome contradicts the subject's expectations based on previous experiences. Although both components within the *anticipation interval* have been associated with a single variable process, the intentional preparation, their scalp distribution suggests a differentiation of these processes. The early component with frontal predominance has been considered to represent primarily the frontal processing of stored contingencies, while the late component has been associated with the preparation for motor or mental performance. Depending upon distinct variations between the experimental designs, the PINV is more pronounced over the frontal or over the precentral cortex. These variations probably induce different cognitive processes, and thereby the different scalp distribution of the PINV. We assume that different PINVs, recorded from different scalp locations, are associated with different cognitive processes. If an analogy between early and late component and PINVs can be drawn, it may be hypothesized that a frontal PINV will develop after changes of expected stimulus propositions without a change of

a planned response to a contingency. These propositions are cues for reinforcers or act as reinforcers themselves. Thus, the frontal PINV may be associated with the preparation for an appraisal of the stored (expected) contingency, whenever an internal model of stimulus contingency and response-outcome contradicts present observations. On the other hand, a precentral PINV, compared to the late SP-component, may signal the preparation for the reappraisal of response propositions, i.e., the evaluation of the adequacy or inadequacy of the learned response (e.g., was the response fast enough). Therefore, the frontally predominant PINV in the present study is attributed to the emphasis on contingency, while the large precentral PINV in the first study may be due to the emphasis on response speed in the instruction. However both processes, response appraisal and contingency appraisal, are usually interwoven. Therefore, a fronto-central distribution of the PINV will be found. Our interpretation is supported by the results obtained from patients with bilateral lesions of the frontal lobes (Elbert et al. 1981). These patients have difficulty in establishing the response-outcome contingency and the adequacy of their response during control trials, although they often manage the response in an objectively successful way. They may have more difficulty in associating their motor response with IS-offset. Therefore, they are less sensitive to the contingency change from control to loss of control, which is reflected by a sharp decrease in PINV amplitude during the second period.

HR responses seem to be sensitive to conditions as experienced by experimental subjects, the acceleration in response to the IS only decreases in control subjects, who show pronounced and even increasing decelerations during the WS-interval. These may be interpreted as a sign of "passive coping" (Obrist 1976) preceding the generally uncontrollable IS. On the other hand, HR data are less consistent with those of previous studies (Rockstroh et al. 1979; Lutzenberger et al. 1981). The differences between the studies may be due to the different samples: an international replication study (Birbaumer, Lang, Elbert, Miller, Lutzenberger, Simons; an attempt at international replication in psychophysiology, unpublished) revealed a strong dependency of HR effects as compared to SPs on a selected subject sample.

In accordance with previous results (Rockstroh et al. 1979; Lutzenberger et al. 1981), SCRs decreased across periods despite the increase in aversive stimulation. Probably, the cognitive processes of contingency or control appraisal are primarily reflected by EEG parameters and are not necessarily associated with autonomic responses.

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