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Evaluation of Contingencies and Conditional Probabilities

A Psychophysiological Approach to Anhedonia*

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Summary. Slow brain potentials, evoked potentials and autonomic responses were investigated in anhedonic subjects and controls. The distribution of physical anhedonia (PA) scores from different samples (students, soldiers, schizophrenics, depressives) is compared. Within a S1-S2 reaction time paradigm, an additional, S2-similar stimulus was introduced during the anticipation interval in 50% of trials (pseudorandom). Subjects had to press the button only to the S2. The additional stimulus (AS) elicits a distinct positive deflection. Anhedonics show larger pre-AS negativity and less reduction in negativity after the S2 (PINV) than controls. The slow wave to S1 as well as the pre-AS negativity vary with the conditional probability of the AS, but to a lesser extent in anhedonics. Anhedonics provide more preparatory negativity prior to and following ambiguous or difficult discrimination tasks, but at the frontal site. Results may suggest impaired contingency evaluation in anhedonic subjects.

Key words: Event-related potentials – CNV – PINV – PCA – Anhedonia – Stimulus discrimination – Depression

Zusammenfassung. Ereigniskorrelierte Potentiale (langsame Potentiale, evozierte Potentiale) und autonome Reaktionen wurden in einem S1-S2-Reaktionszeitparadigma bei Personen erhoben, die auf einer Anhedonieskala hohe oder niedrige (Kontrollgruppe) Werte aufwiesen. Die Verteilung der Anhedoniewerte wurde für verschiedene Gruppen (Studenten, Soldaten, Schizophrene, Depressive) verglichen. Während des sechssekündigen Antizipationsintervalles wurde in 50% der Durchgänge (zufällig) ein dem imperativen Reiz ähnlicher Reiz dargeboten; die Probanden sollten aber nur auf den S2 hin mit Knopfdruck reagieren. Bei Probanden mit hohen Anhedoniewerten zeigt sich gegenüber Kontrollpersonen eine stärkere negative langsame Potentialverschiebung vor dem Zeitpunkt des zusätzlichen Reizes (AS) sowie ein geringerer Rückgang der Negativierung nach dem S2 (PINV). Die

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Negativierung vor dem AS sowie die „slow wave" auf den S1 variieren mit der bedingten Wahrscheinlichkeit des AS, jedoch weniger deutlich bei Anhedonikern. Der AS selbst löst bei allen Probanden eine positive Verschiebung aus. Die Ergebnisse lassen eine beeinträchtigte Fähigkeit zur Kontingenzermittlung bei Anhedonikern vermuten.

Schlüsselwörter: Ereigniskorrelierte Potentiale - CNV - PINV - PCA - Anhedonie - Zusätzliche Reize - Depressionen

Introduction

One key to the understanding of psychiatric disorders is the study of subjects susceptible for psychopathological developments. It is assumed that the determination of high risk factors enlightens those factors which may contribute to the development of the disorder. Based on Meehl's (1962) concept of schizotypy, Chapman and co-workers (Chapman et al. 1976 and 1978) developed a self-report questionnaire to disclose physical and social anhedonia, for the selection of prepsychotic non-clinically conspicuous schizotypes. Anhedonia is thought to reflect a major potential risk factor toward a psychiatric disposition, particularly for the development of a schizophrenic disorder. A genetic predisposition to anhedonia has been suggested by neurohistochemical findings, which had been interpreted as distortion of subcortical reinforcement mechanisms, brought about via a 6-hydroxydopamine excess (Stein and Wise 1971; Wise and Stein 1973).

One scale (35 items) of the Chapman questionnaire, which was adopted for the present study, involves "perceptual aberration or body image distortion", while the other scale, "physical anhedonia", describes a deficiency in the experience of pleasure, especially to physical stimuli (61 items). (A lie scale consists of another 17 questions). "The anhedonia which we wish to measure is a life-long characterological defect in the ability to experience pleasure. Pleasure is characterized by a strong positive affect, by a keen anticipation of the experience that evokes it, by a satisfying recollection of the experience, and by a willingness to expend effort to achieve the experience. Behaviors which evoke pleasure tend to be repeated" (Chapman et al. 1976, p. 376).

According to Chapman et al. one-third of schizophrenic patients can be considered anhedonic according to the questionnaire scores, whereas depressives seem to lack a consistent relation. However "full-blown deep depression as in depressive psychosis might, nevertheless, affect scores on the scales" (Chapman et al. 1976, p. 379). We reveal considerably high physical anhedonia (PA) scores in hospitalized depressives (Fig. 1), which questions the concept of PA as being primarily indicative for a schizotypic personality. The database obtained up to now is certainly too small to allow any definite conclusions about significance and limits of the scales to discover risks for psychopathological developments; this must await long-term follow-up of the samples.

The present study addresses further clarification and specification of the difference in response patterns between anhedonic subjects selected according to PA scores- and hedonic controls. Therefore we briefly outline the differences reported so far:

Subjects scoring deviantly high on the PA scale have been found to exceed controls on Rorschach indicators of schizophrenic-like thought disorders (Edell and Chapman 1979), and to show impaired social skill, as measured by a role-playing test (Haberman et al. 1979). No correlation between the PA scores and social adjustment however, could be documented ($r < 0.2$), when we interviewed the present sample, using Chapman's inventory (Weissman and Paykel 1974) (translated into German). No significant relation to ratings on a psychoticism interview scale was found in our samples. On the other hand, Chapman et al. (1980) reported more schizotypic symptoms, greater social isolation, and lower heterosexual interest and activity for PA-high subjects, than for controls.

Furthermore psychophysiological investigations clearly exhibit different psychophysiological response patterns for groups with high versus low scores on the questionnaire scales (Lutzenberger et al. 1981a; Simons 1981 and 1982; Miller 1981; Simons et al. 1982; Miller et al. 1981; Ward et al. 1981). Using a standard procedure, Simons (1981) observed deviant heart rate responses and reduced skin conductance responses (SCR) in anhedonics, comparable to those reported by Venables 1975; Gruzelier and Venables 1972 and 1973) for schizophrenic subgroups (hyporesponders). No SCR reduction became obvious in a signal detection task. A reduced sensitivity to positive reinforcement, as indicated by SCR amplitude and the second deceleration of the anticipatory heart rate response, confirms and extends the characterization of the anhedonic group (Lutzenberger et al. 1981b).

Increasing attention has been devoted to event-related slow brain potentials, since they reflect preparation and information processing. A slow negative potential shift that develops in anticipation of a signalled event, to which a distinct decision or response is required, is considered to represent cerebral preparatory potentiality for the performance required with the signalled, or imperative, event. The slow potential (SP) components are sensitive for variables such as contingency, response requirements, consequences etc. (for review see Rockstroh et al. 1982). In anhedonics we observed a smaller resolution of slow contingent negative brain potentials [or an elevated post-imperative negative variation (PINV) respectively] than in controls, whenever ambiguity or uncontrollability was introduced into the experimental conditions (Lutzenberger et al. 1981a). In line with Giedke and Bolz (1980) and Delaunoy et al. (1978) we interpreted the PINV as reflecting an inability (possibly listlessness) to resolve uncertainty, brought about by deficient evaluation of the contingencies. (Contingency means the association or connection of events, i.e., stimuli, responses, consequences, due to their spatial and/or temporal contiguity. Contingency evaluation is the prerequisite for conditioning, attribution, and control; see Seligman 1975).

A PINV has also been found in healthy volunteers, when control over an aversive stimulus was unexpectedly withdrawn (Rockstroh et al. 1979; Elbert et al. 1982). It seems noteworthy that a prolonged PINV has been reported for psychiatric patients, especially schizophrenics and endogenous depressives (Dongier et al. 1976 and 1977; Abraham et al. 1980).

The Madison group (Miller 1981; Miller et al. 1981; Simons 1981b) report a reduced P300 in anhedonic subjects, as compared to controls, under different signal detection conditions. The P300, a positive deflection of the event-related

potential (ERP), following 300–400 ms after the signal stimulus, is attributed to the evaluation of the stimulus significance and to storage (memory) processes of the event (Rockstroh et al. 1982).

All these results point to the sensitivity of ERPs (especially those related to contingency appraisal) in psychosis-vulnerable subjects. Therefore the present study was designed to clarify the effects of contingency change and ambiguity reflected by ERPs on anhedonics. If, above all, the appraisal of contingencies is impaired in psychosis-vulnerable subjects, then these should exhibit larger slow negative shifts as signs for increased preparational effort for the contingency assessment; although accompanied by poorer performance. The less effective stimulus selection should be reflected by a reduction in the corresponding ERP components. The present study tested these hypotheses by occasionally presenting an additional, S2-similar stimulus within the S1–S2 interval. The need for stimulus discrimination introduces uncertainty.

The Distribution of PA Scores

The distribution of the scores on the PA scale for the German translation of Chapman's questionnaire is presented in Fig. 1 for several samples.

A total of 750 Tübingen University students filled in the questionnaire. The distribution of the male sample ($n=463$, mean PA score: $11.3+/-7.0$) differs from that of the females (mean PA score: $8.8+/-5.0$, $P< 0.01$ for the sex difference) in that the latter lacks scores higher than 20 (including only 1.7% of the sample, as compared to 9.8% for the males). No age dependency is observed for the PA score in the samples: mean age of the male students was 24 years, with a range of 20–31 years. (Correlation PA/age $r=-0.06$, N.S.)

Although a tendency for increased PA scores was established in a sample of 230 German soldiers (mean PA score: $13.0+/-7.0$, mean age 21 years) (Merz 1982), distributions are quite comparable (see Fig. 1).

PA scores of hospitalized schizophrenics exceed those of the student and soldier samples. However, a remarkable number of patients do not exhibit any anhedonic tendencies (Fig. 1). In contrast, 19 out of 20 depressives gave a strong anhedonic self-report. (Mean PA for schizophrenics: $15.9+/-8.7$, mean age 34 years, range 22 to 64 years; mean PA for depressives: $22.9+/-6.9$, mean age 51 years, range 28 to 76 years; as for the other samples no significant relationship between age and PA score was found.)

Thus, the distribution of PA scores for the different samples does not strengthen the view of PA being differentially predictive for the schizophrenic syndrome. Depression hardly lacks PA as measured by the self-report scale.

Method

Subjects. Male student volunteers ($n=38$) were selected out of the student population. For the present study 22 subjects with PA scores larger than 15 were assigned to the anhedonic group, 16 subjects with scores below 10 constituted the control group (see Table 1 for means and re-

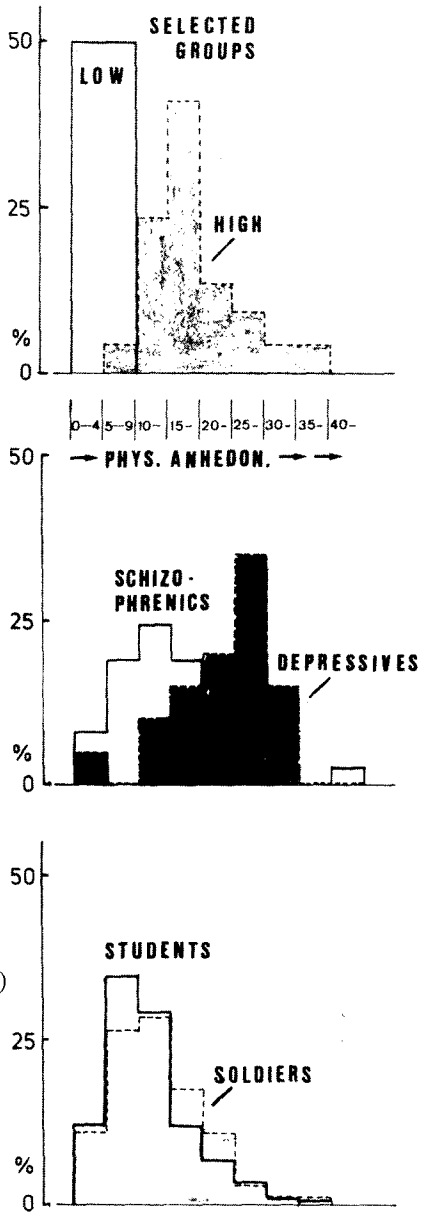

Fig. 1. Relative frequency distributions (in percent) of the PA score. Subjects filled in the German version of Chapman's questionnaire: *Upper graph:* 16 students with low scores *(light)* and 22 students with high scores *(dark)* were selected for the present study. Middle graph: 37 schizophrenic *(light)* and 20 depressive *(dark)* inpatients. *Lower graph:* 463 students *(light)* and 230 soldiers *(dark)*

Table 1. PA scores for the two selected groups during three evaluations

	July 1978	March 1979	September 1980
PA-high	21.9	19.0	17.3
Controls	5.7	5.4	4.8

The PA score decreases across the repeated evaluations ($F(2/64)=4.3$, $P<0.05$)

ference values).[1] It was assured that subjects were not under current medication and had not suffered from any cardiovascular or CNS abnormality. Subjects were paid 30 DM (about $ 15 U.S.A. currency) for their participation in the experimental session which lasted for about 1 h.

Design and Procedure. Within the two-stimulus anticipation paradigm, each trial started with a sound stimulus (white noise of 80 dB SPL for 100 ms) as S1. After a constant interstimulus interval (ISI) of 6 s, a white noise of 80 dB was presented as S2. Subjects were instructed to interrupt the S2 by pressing a button as quickly as possible. The 80 dB white noise would terminate with the motor response. In 50% of the trials, a very similar noise (white noise of 80 dB, filtered for frequencies above 1 kHz) appeared during the interval 4.0–4.2 s of the ISI. Within the total 50 trials, trials with this additional stimulus (AS) and trials without AS were presented in random order.

Subjects were instructed that an AS similar to the S2 would be presented in some of the trials, but that their task was only to respond to S2. Thus, the discrimination in time and quality between AS and S2 was required. During the experiment, subjects sat in a reclining chair in a partially electrically shielded, dimly lit, sound-attenuated room. Subjects held the microswitch in their dominant hand. After electrode placement subjects read task instructions and were introduced into stimulus qualities and response requirements by 4 practice trials (with and without AS). Practice trials were supervised by the experimenter to make sure that all subject were familiar with the response requirements. Subjects were explicitly asked to visually fixate any convenient point in front of them throughout the experiment and to avoid any movement of head and eyes.

Apparatus and Physiological Recordings. A PDP 8/e laboratory computer controlled the timing and the presentation of all stimuli, and stored the physiological data and response latencies on magnetic tape. Pressing the microswitch closed the Schmitt trigger in the PDP 8/e so that the response latency was stored to the nearest 1 ms. The EEG was recorded from frontal (Fz), precentral (Cz) and parietal (Pz) leads, and from both precentral hemispheres (C3, C4), referred to shunted earlobes, with a time constant of 30 s. Grass flat silver disc electrodes, chlorided before each experimental session, were used; Grass EC2 paste served as the conducting agent. The electrode 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 electrode impedance levels of less than 10 k Ω Earlobe electrodes were held in place with clips. A calibrator provided a 50 μV square wave pulse to the EEG channels 5 s after each S2. EEG activity was amplified by a Beckman type 9806 AC 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 and collapsed to 100 ms points by a digital filter without phase distortion for the slow wave analysis.

Vertical and lateral eye movements (VEOG, LEOG) were recorded, amplified, and processed in the same manner as the EEG channels, except for the use of Beckman Ag/AgCl electrodes which were attached above and below the left eye (VEOG) or the nearest of the outer canthi (LEOG) respectively.

A bipolar recording of the ECG from leads V1 (right sternal margin at the 4th intercostal space) and V5 (left anterior axillary line–5th intercostal space) was fed into a cardiotachometer coupler to detect the QRS waves, which triggered the computer clock. The interpulse interval data were subsequently converted to a rate-per-sec format, each beat being weighted by the proportion of the second that it occupied.

Data Reduction and Analysis. Trials were excluded if a mean deviation in one of the EOG channels exceeded 20 μV. This resulted in the exclusion of 12 out of 50 trials on an average. The resulting average EOG responses were small (see Fig. 2), and no statistical effects were found.

1 The present study is part of a larger investigation on subjects selected according to both scales of the Chapman questionnaire. Further results are reported in Lutzenberger et al. (1981a and b)

The physiological data were averaged over trials separately for each subject × condition × recording cell. To define SP parameters, principal component analyses (PCA) were computed A) for the 4-s interval between S1 and AS, B) for a 4-s interval starting with the AS and terminating 2 s after the S2. Components of the evoked potentials (EPs) during a 450 ms interval following each stimulus (S1, AS, S2) were also determined by PCA. The covariance matrices used were normalized by equating the trace with its dimension. The number of components retained was set at the number of "eigenvalues" equal to or greater than unity. Components were rotated using a normalized varimax solution. A least square fit was performed to obtain the component scores (factor scores) as measures of the contribution of each of the components to the individual brain wave. The linear combination of the components is fitted to each potential course.

While analysis B) takes into account the effects of the AS presentation, analysis A) covers possible effects of the experience of the preceding trial that may have influenced the expectancy of an AS or no AS to come and, thus, the physiological responses prior to the AS. The conditional probability of two subsequent identical trials (AS–AS or no AS–no AS) was only 1 in 3, whereas the probability of a change from AS trial to no AS trial or vice versa was 2 in 3. Subjects obviously take these different probabilities into account, in that they establish subjective probabilities regarding the next trial on the basis of the preceding one. Following a trial with AS, a subsequent trial without AS is more likely than another trial with AS, while subjects will expect a trial with AS to follow a trial without AS.

Effects were statistically evaluated by analyses of variance (ANOVAs) computed separately for the recordings along the midline (Fz, Cz, Pz) and for the central recordings (C3, Cz, C4), with the between-subjects factor GROUPS (anhedonics versus controls) and the within-subjects factors CONDITION (trials with AS versus trials without AS—analysis B) only), PRECEDING TRIAL EXPERIENCE, PR (comparison of trials following a trial with AS and trials following a trial without AS—analysis A only), and TOPOGRAPHY (Fz versus Cz versus Pz, or C3 versus Cz versus C4, respectively).

Results

Effects of the Presentation of the AS and of the Preceding Trial Experience on

A. Reaction Time. All subjects respond faster to the S2, if this S2 was preceded by an AS as compared to trials without AS. The mean difference of 189 ms between the conditions (210 vs 399 ms) gives rise to a main effect of CONDITION with $F(1/36)=122.0$, $P< 0.001$.

B. Slow Brain Potentials. Following the S1 a negative shift develops in all recordings (see Fig. 2). Figure 2 also illustrates a marked reduction in negativity that is produced by the presentation of an AS (solid line in Fig. 2), which is, however, compensated by the generation of a subsequent negative shift prior to the S2.

Grand averages and principal components, defined by the PCA for the 4 s S1-AS interval (analysis A)), are given in Fig. 3a; grand averages and components for analysis B), i.e., the 4 s interval starting with the AS are illustrated in Fig. 3b.

The component structure obtained for the 4 s interval following the S1 is remarkably similar to the structures obtained from different designs, including S1–S2 intervals of different length. This is illustrated in Fig. 3a: the dotted lines represent components reported by Lutzenberger et al. (1981c) for a series of studies using 6 s anticipation intervals. In addition to the three components, labelled "early", "mid", and "late", a component contributing in the very beginning of the S1 interval is obtained which can be compared with the "slow wave„

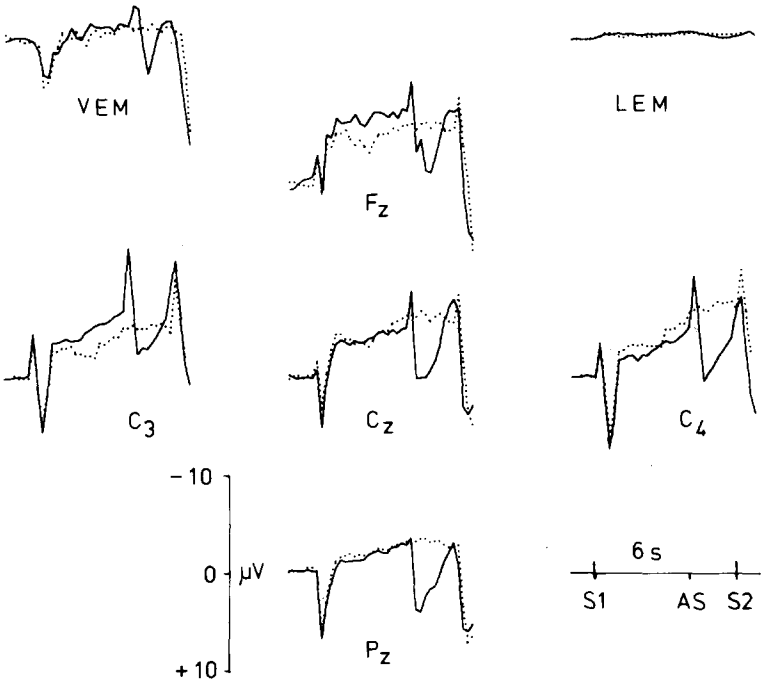

Fig. 2. Across subjects average for the SPs and eye movements *(VEM, LEM)*, separated for trials with AS *(solid line)* and without AS *(dotted line)*

(SW) in EP analyses (see also section c)). Correspondingly, the SW scores are positive, with centro-parietal dominance (main effect of TOPOGRAPHY with $F(2/72)=24.9$, $P<0.01$ for the Fz-Cz-Pz ANOVA).

The early component contributes to negative values at Fz but to positive values at Pz (main effect TOPOGRAPHY with $F(2/72)=26.3$, $P<0.01$, see also Fig. 4). The fronto-central gradient tends to be affected by the preceding trial experience, being less pronounced when an AS had occurred in the trial preceding the actual one, in which this gradient is measured: interaction TOP × PR with $F(2/72)=2.5$, $P<0.1$ for Fz-Cz-Pz analysis.

The strong frontal negative shift is also reflected by the middle component (TOPOGRAPHY: $F(2/72)=5.3$, $P<0.01$ for Fz-Cz-Pz), whereas no significant TOPOGRAPHY effects are obtained for the late component scores.

The late component prior to an AS is significantly larger, if the preceding trial was without AS, as compared to trials following an AS trial. This is documented by a main effect of PR with $F(1/36)=5.8$, $P<0.05$ for the Fz-Cz-Pz ANOVA, and $F(1/36)=12.5$, $P<0.01$ for the C3-Cz-C4 ANOVA. Thus, high expectation of an AS to be presented induces larger preparatory negative SP shifts than low expectation (see also Fig. 5).

A component with maximum loadings at about 0.7 s following the AS is labelled "AS-positivity", since this component is only prominent after AS presentation, thus describing the decrease in negativity or a positive shift, respectively: main effect of CONDITION: $F(1/36)=56.5$, $P<0.001$ for the Fz-Cz-Pz ANOVA,

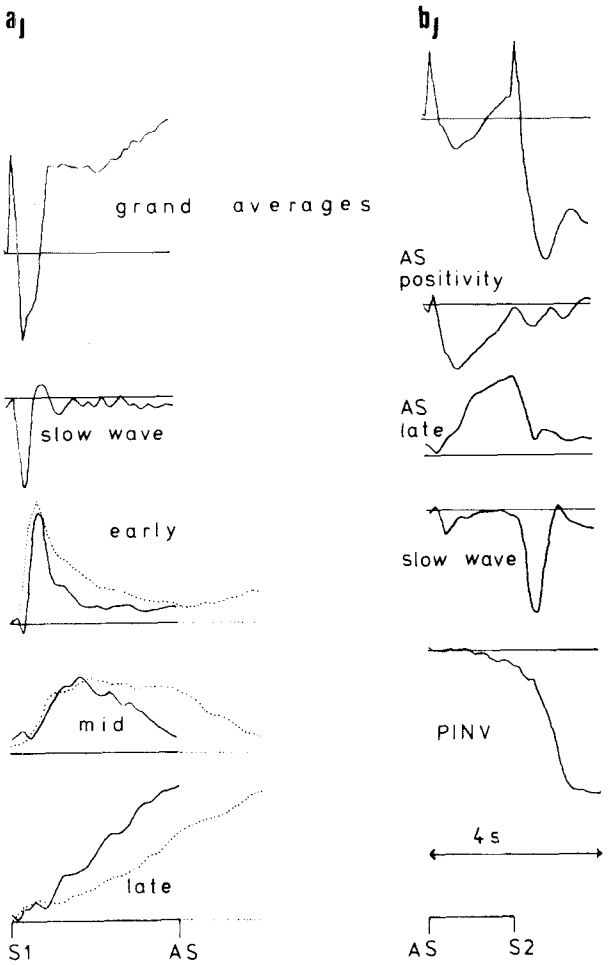

Fig. 3a and b. PCA. **a** Covering the 4 s interval between *S1* and *AS (left column)* and for analysis **b** *(right column)* which includes 2 s *AS–S2* and a 2 s post-*S2* interval. The dotted lines represent components for comparable designs with using 6 s intervals (see text). The polarity of components was chosen according to their averaged component scores (negativity is up)

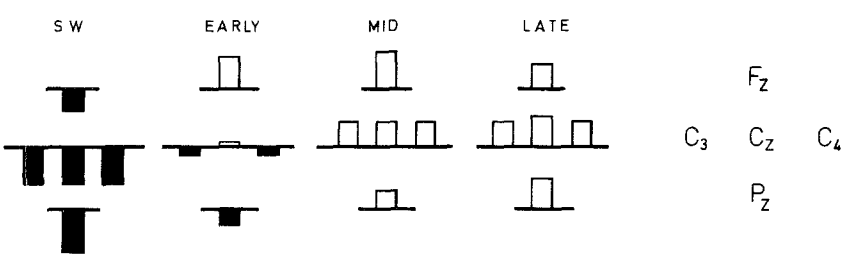

Fig. 4. Topographical distribution of the PCA B). Component scores are normalized according to the component's contribution at its peak loading of the corresponding wave form

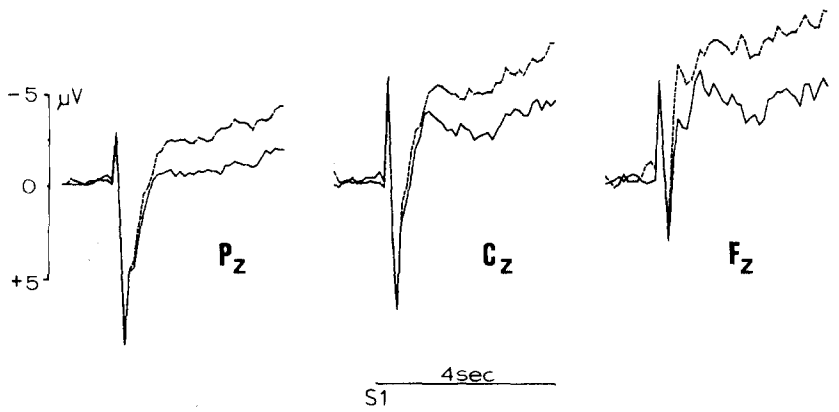

Fig. 5. SPs are higher, when the actual trial had been preceded by a trial without AS, i.e. when the objective probability of an AS to be presented is 2 in 3 for the actual trial (Prec trial with AS = ——; Prec trial without AS = ------)

$F(1/36)=39.7$, $P<0.001$ for the C3-Cz-C4 ANOVA. For the lateral comparison (C3-Cz-C4), a main effect of TOPOGRAPHY with $F(2/72)=7.8$, $P<0.05$, as well as an interaction of TOPOGRAPHY × CONDITION with $F(2/72)=25.9$, $P<0.01$, document the larger positive deflection at the vertex as compared to C3 and C4.

The negative shift subsequent to this AS-positivity, peaking at S2 onset, is described by the "AS late" component. This late component is most pronounced at the vertex, as is documented by a main effect of TOPOGRAPHY for all recordings: Fz-Cz-Pz: $F(2/72)=3.3$, $P<0.05$; C3-Cz-C4: $F(2/72)=3.8$, $P<0.05$. For the lateral recordings, an interaction of CONDITION × TOPOGRAPHY ($F(2/72)=9.9$, $P<0.01$) can be attributed to the fact that following an AS, the most pronounced late negative shift develops at the vertex (−9 μV), as compared to smaller shifts at C3(−4 μV) and C4 (−2 μV), while nearly no differences between the recordings are found in trials without AS.

As reported for the S1 response, the S2 is followed by a component labelled slow wave (SW), which shows transient loadings with maximum in the range of 0.4 to 0.6 s following the S2. The SW contributes to positive values at Cz and Pz, but exhibits zero to negative component scores at Fz. This scalp distribution gives rise to a main effect of TOPOGRAPHy for Fz-Cz-Pz with $F(2/72)=4.6$, $P<0.05$. However, the fronto-parietal gradient is markedly diminished if an AS had preceded the S2: interaction TOPOGRAPHY × CONDITION with $F(2/72)=8.1$, $P<0.01$ for Fz-Cz-Pz. Thus, following an AS, the SW contributes equally positive along the midline, while in trials without AS, this positive shift to S2 shows a characteristic fronto-central gradient with positive parietal maximum and small negative values at Fz.

Finally, the resolution in anticipatory (pre-S2) negativity is described by the component PINV (post-imperative negative variation or S2-positivity, see Figs. 2 and 3). This resolution is smaller precentrally in trials with AS as compared to trials without AS: $F(1/36)=5.3$, $P<0.05$ for the C3-Cz-C3 analysis.

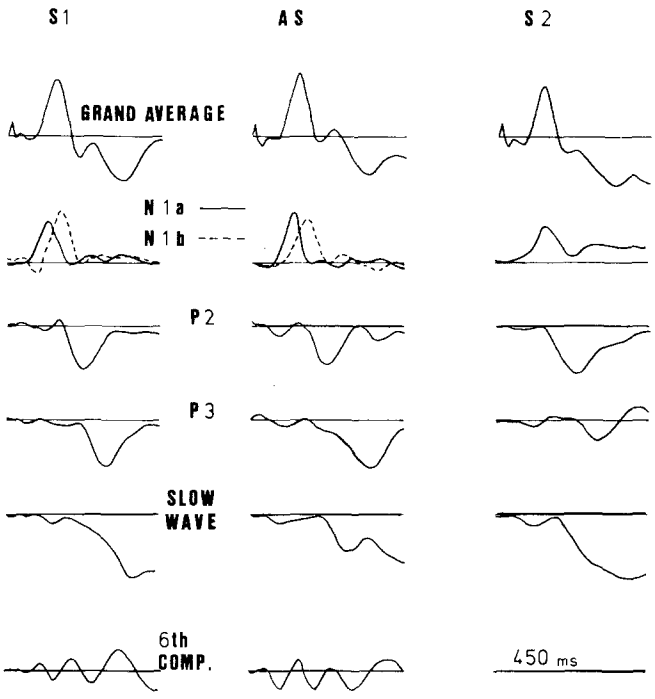

Fig. 6. PCA of the EPs in response to *S1, AS* and *S2,* each computed for an interval of 450 ms following the stimulus

C. Evoked Potentials. The PCAs of the EPs, determined for 450 ms intervals following the S1, the AS, and the S2 result in similar component structures (see Fig. 6).

The basic principal components labelled N1a, N1b (N100 scores), P2, P3 (P300 score), and SW are repeatedly reported for acoustic EPs. Whereas the component N1a peaks at 120–130 ms i.e., prior to the N100 peak of the average EP (150 ms), the N1b (170–180 ms), shows maximum loadings after the N100 peak. The latter principal component is not extracted for the S2 EP (see Fig. 6). This is due to the extraction criteria applied in the present study: Since the resolution of negativity increases the variance of the positively contributing components considerably, the relative N1b variance decreases in turn, so that the corresponding "eigenvalue" is too small.

The N1a is larger at C3 than at C4 in response to the AS (TOPOGRAPHY $F(1/36)=13.9$, $P< 0.01$) and in response to the S2 (TOPOGRAPHY $F(1/36)=16.2$, $P< 0.01$), but does not differ significantly when evoked by the S1. Instead, the S1-elicited N1b exhibits left-hemispheric (C3) dominance: $F(1/36)=4.6$, $P< 0.05$, as does the S1-elicited SW ($F(1/36)=14.3$, $P< 0.01$). The P2 evoked by the S2 is also larger at C3 than at C4: $F(1/36)=9.3$, $P< 0.01$).

If no AS had occurred in the preceding trial (and may therefore be expected in the current trial), the P3 is larger in response to the AS, as compared to the P3

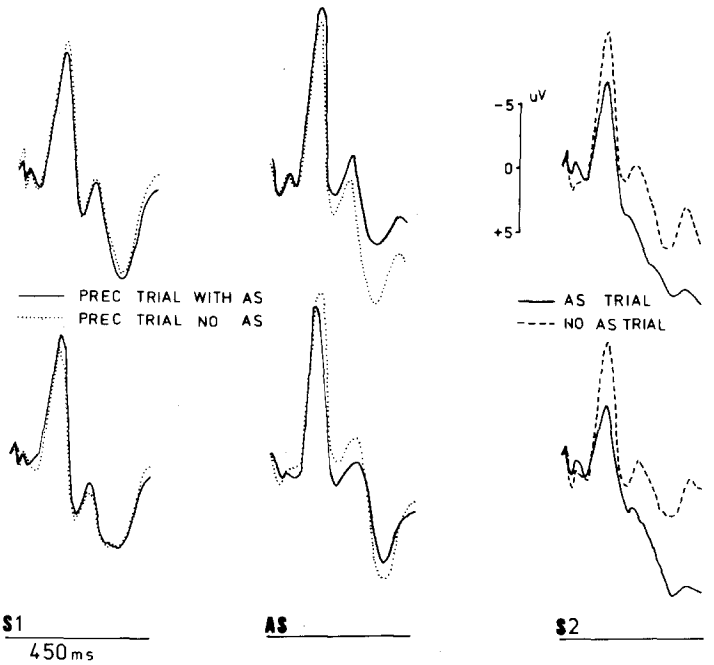

Fig. 7. EPs response to the acoustic *S1, AS,* and *S2* for controls *(lower traces)* and subjects with high scores on the PA sclae *(upper traces)*

following less expected ASs (in trials following an AS trial); this is documented by a main effect of PR with $F(1/36)=6.2$, $P< 0.05$ (see Fig. 7).

Further effects of the preceding trial experience are different for the two experimental groups, and are therefore reported below.

The presentation of an AS changes the S2 EP (see Fig. 7), in that the N1a is reduced (main effect of CONDITION: $F(1/36)=19.5$, $P< 0.01$), and no P3 can be detected in the average course ($F(1/36)=5.8$, $P< 0.05$). On the other hand, SW scores are about twice as large in trials with AS ($F(1/36)=9.4$, $P< 0.01$), this difference being more pronounced at C3 than at C4 (interaction of TOPOGRAPHY × CONDITION: $F(1/36)=19.2$, $P< 0.01$).

D. Autonomic Responses. Mean heart rate is larger in trials with AS as compared to trials without AS (main effect of CONDITION with $F(1/36)=13.2$, $P< 0.001$). The same is found for SCR, being higher in AS trials as compared to no-AS trials ($F(1/36)=9.9$, $P<0.01$). Thus, autonomic responses point to higher autonomic arousal under the condition of increased discrimination requirements. The triphasic pattern of the heart rate (deceleration–acceleration–deceleration, scored according to Gatchel and Lang 1973) does not show effects of the PRECEDING TRIAL EXPERIENCE; neither do SCR amplitudes. The presentation of an AS reduces the amplitude of the second heart rate deceleration (main effect of CONDITION: $F(1/36)=28.4$, $P< 0.01$), as well as the subsequent acceleration ($F(1536)=4.2$, $P< 0.01$).

Table 2. Resonse speed in ms

S2 preceded	PA-high	Controls
By an AS	265	284
By no AS	428	451

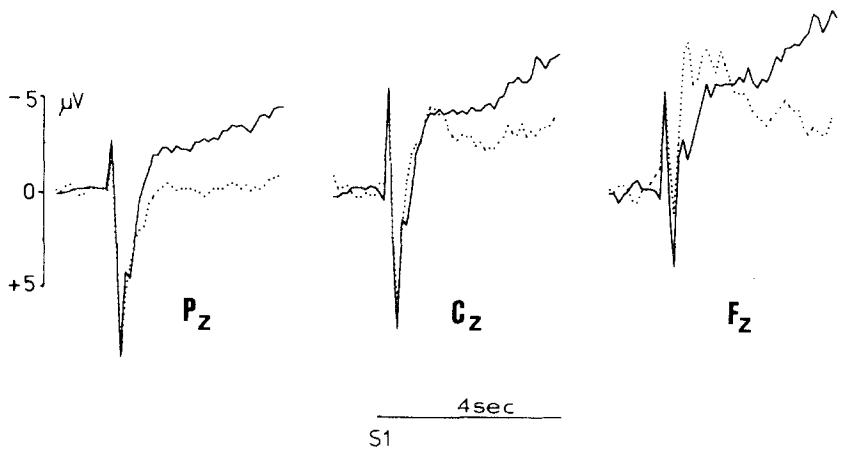

Fig. 8. SPs for the S1-AS interval averaged separately for anhedonics *(solid line)* and controls *(dotted)*. Note the reduced gradient along the midline of the early negativitly and the overall elevated negativity towards AS in anhedonics

Differences between Anhedonics and Controls with Respect to

A. Reaction Time. No significant group effects are demonstrated for response speed.

B. Slow Brain Potentials. For the middle component within the S1-AS interval, an interaction of GROUPS × TOPOGRAPHY ($F(1/36)=3.8$, $P<0.05$) points to the more pronounced fronto-central gradient in control subjects (component scores are 5:3:1 versus 2:2:2) as compared to anhedonics (see Fig. 8).

The late component within the S1-AS interval is larger in anhedonics than in controls, as in documented by a main effect of GROUPS with $F(1/36)=5.6$, $P<0.05$ for the C3-Cz-C4 analysis.

The SW in response to the S1 shows an interaction of GROUPS × PR ($F(1/36)=4.2$, $P<0.05$ for the Fz-Cz-Pz ANOVA, and $F(1/36)=3.1$, $P<0.1$ for the C3-Cz-C4 ANOVA), indicating that control subjects exhibit the smaller SW in trials following a trial without AS as compared to a trial with AS, while anhedonics are less sensitive for the preceding trial experience with respect to the SW component.

Groups differ, furthermore, with respect to the post-S2 negativity resolution (the component PINV), in that anhedonics show the smaller resolution (i.e. the larger PINV), when the AS had been presented than following its omission. Control subjects, on the other hand, tend to show no difference between the con-

ditions: GROUPS × CONDITION: $F(1/36)=3.9$, $P< 0.1$ for C3-Cz-C4; $F(1/36)=8.3$, $P< 0.01$ for Fz-Cz-Pz.

Taken together, anhedonics show the larger preparatory negativity prior to an AS and less negativity resolution following the S2 in AS trials, as compared to controls. The usually observed frontal dominance of the early to middle negativity is clearly diminished in anhedonics (see Fig. 8).

C. Evoked Potentials. In response to the S1, control subjects show the smaller N1b in trials following a trial without AS as compared to trials following a trial with AS, while (similar to the above noted SW results) nearly no sensitivity to the preceding trial experience is shown by anhedonics (see Fig. 7); this is documented by the interaction of GROUPS × PR with $F(1/36)=4.2$, $P< 0.05$.

On the other hand, the N1b elicited by the AS is larger in controls when the AS was expected to occur (i.e., in trials following a no-AS trial), than when AS expectancy is low. This difference in N1b amplitudes tends to reverse in anhedonics (see Fig. 7), giving rise to an interaction of GROUPS × PR with $F(1/36)=6.2$, $P< 0.05$.

The left-hemispheric dominance of the S2-evoked N1a (as described above) is more pronounced in anhedonics than in controls: GROUPS × TOPOGRAPHY: $F(1/36)=4.9$, $P< 0.05$.

D. Autonomic Responses. No group differences are documented for heart rate and SCR.

Discussion

Regulation of the Slow Brain Potential

Describing a model of SP regulation, we have stated that slow negative shifts indicate preparatory potentiality for cerebral performances (Rockstroh et al. 1982). The negativity is considered unspecific with respect to the type of performance or response anticipation, but develops specificity with respect to a spatial distribution, in that it is generated in those brain regions needed according to the subjective prediction. This prediction, which is derived from contingency models involves activity of the frontal cortex (in the S1–S2 paradigm usually reflected by the early component) which in turn regulates SPs of other cortical regions (= late component) via a thalamo-cortical feedback-loop (based upon the neurophysiological model of Skinner and Yingling 1977). Thus late negativity indicates nothing else than a preparedness of brain regions for anticipated requirements. The performance or processing by itself consumes the potentiality; correspondingly, a decrease in negativity or a positive shift, respectively, is observed (provided no need for simultaneous generation of additional potentiality is evaluated by the subject).

Such a relative positive shift is generated by the additional stimulus, to be processed during the anticipation interval. The task of discrimination of the response-inhibiting AS and the response-requiring S2 consumes cerebral potentiality. As predicted from the SP regulation model this performance is accompanied by the positivity. Permanent reduction in negativity, which results from

distracting stimuli (Tecce 1972; Tecce et al. 1978 and 1982), presented throughout the S1–S2 interval can be considered to result from the very same mechanisms. The present results indicate processing of an additional stimulus as cause of the reduction in negativity, which is hardly attributable to divided attention (Tecce 1972), since the AS is task-relevant in that it requires inhibition of the motor response. In line with the SP regulation model, further potentiality/negativity is provided subsequent to the processing of the AS, for the anticipated requirements at S2 (motor response, evaluation of response-contingencies etc.).

When an AS had preceded the S2, recognition of S2 is easy and selective attention may be low. In accordance with Hillyard and Woods (1979) the N100 is reduced under this condition. Furthermore, the S2-elicited potential lacks a P300, if and only if an AS had preceded. We may explain this result, if the P300 is considered a manifestation of a process whereby schemata are revised (Donchin 1981): after AS-recognition, the S2 is predicted, and the trial can be correctly categorized. The S2 then merely may confirm the schema and no P300 is elicited. If no AS had preceded, schemata can be restructured though not before S2 presentation.

Furthermore, there is evidence that the preceding experience of experimental conditions affects expectancies regarding the actual or anticipated conditions, and thereby ERPs: Following a trial with AS, the conditional probability of another AS occurring has been only 1 in 3. The P300 covaries with these probabilities, in accordance with the results of Johnson and Donchin (1980 and 1982). If an AS is expected, more preparatory potentiality is provided, reflected by markedly larger negative shifts prior to the AS (Fig. 5). Thus, the present SP data confirm, and may have been predicted, from the SP regulation model, although further specification and elaboration is necessary to derive quantitative relations, e.g., to predict SP levels when performance includes different dimensions. Examples are motor commands, stimulus processing, contingency and response-outcome evaluations (Rockstroh et al. 1982). None of these factors is a necessary prerequisite to elicit (late) negative SPs; e.g. a late component is observed prior to the AS which did not ask for an overt motor response. The overall negativity prior to the AS shows frontal dominance, indicating preparational processes of frontal contingency comparators, whereas a precentral maximum prior to the S2 confirms that motor requirements prepare primarily somatosensory areas. A marginally higher negativity at C3 than at C4 may be explained by the right-hand response.

The hemispheric asymmetry of motor commands may also be a source for left/right differences observed for the evoked potentials.

Deviances in Anhedonics

A delayed and less frontally located early negativity in anhedonics (Fig. 8) tempts us to speculate that anhedonics possibly lack highly effective frontal contingency processors, located in the frontal and prefrontal cerebral cortex (Luria 1961 and 1973; Loveless 1979). Anhedonics may try to compensate by an increase in potentiality, as exhibited by the PA-high group under the present experimental con-

ditions. It seems interesting to note that increased cortical negativity is not a general feature of the present PA-high sample, but specific for the present task (e.g. no CNV differences or even tendencies to reduction have been observed under different experimental conditions; Lutzenberger et al. 1981a). N100 differences fit with these considerations, in that anhedonics as compared to normals exhibit less sensitivity to the preceding trial experience in N1b scores, elicited by the S1, and by the AS. Furthermore, anhedonics showed the less pronounced reduction in negativity following the S2/response in those trials in which an AS had occurred. Do these tendencies to PINV-like responses again point at an uncertainty of contingency evaluation? A deficit in discrimination also seems to be a possible explanation; however, anhedonics are as fact as controls in pressing the button. Certainly different views are possible. A deficit in resolving experimental ambiguities, i.e., in the evaluation of complex and changing contingencies for anhedonics has been suggested by quite a number of previous results (see Introduction, Miller 1981; Lutzenberger et al. 1981a and b).

But the data do not document causal relations. Is it that anhedonics are less sensitive to the pleasure of detecting contingencies, relationships? Or is it an impaired cognitive ability, which prevents them from successful detection, while anhedonia then results from repeated experiencies and feelings of uncontrollability, due to the misinterpretations (following Seligman's theory for the genesis of depressive-like states, 1975).

We may conclude that both an insensitivity for the rewarding aspects of curiosity (Unlust zur Neugierde) and the inability of contingency evaluation (Unfähigkeit zu Erkennen), are intimately interwoven, favouring each other, finally leading to the risk of a psychotic development!

Following Seligman's concept, endogenous depression is most likely to develop. Thus, we suggest that the PA score is among the potential risk factors for *depressive* development. Addional support is provided by

- the distribution of the PA scores for inpatients (Fig. 1);
- the deficit in right-hemispheric S2-elicited N100, which parallels the finding of Perris and Monakhov (1979), of decreased right-hemispheric synchronization in depressives;
- the PINV in response to withdrawal of control (Elbert et al. 1982), and in PA-high subjects (Lutzenberger et al. 1981a);
- the insensitivity of PA-high subjects for reward as suggested by autonomic responses (Lutzenberger et al. 1981b).

This questions the view of PA being predictive for schizophrenic-like disorders, "rather schizophrenia could promote anhedonia".

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