Sensation Seeking and Affective Disorders: Characteristics in the Intensity Dependence of Acoustic Evoked Potentials

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Key Words

Bipolar affective disorder · Unipolar affective disorder ·
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Abstract

Augmenting/reducing of the evoked potential has been shown to be related to sensation seeking (SS) and specific clinical disorders. Buchsbaum demonstrated that patients with bipolar affective disorders (BAD) tend to be augmenters, as is the case with sensation seekers, and patients with unipolar affective disorders (UPD) tend to be reducers. In addition, he reported that prophylactic medication reduced the tendency to augment in bipolar patients. However, evidence for these relations is restricted to a few studies. This study explores whether Buchsbaum's initial findings can be found in a naturalistic clinical setting. Acoustic evoked potentials were recorded for six levels of intensity (59, 71, 79, 88, 92, 96 dB SPL) from 24 healthy adults, 21 unipolar depressed patients, and 21 patients with BAD. Participants also completed personality questionnaires, especially the Sensation Seeking Scales Form V. Results revealed a positive correlation between SS and augmenting/reducing in healthy controls, thereby replicating earlier findings. Bipolar depressed patients showed larger P1/N1 slopes than healthy controls, when medication was statistically controlled. Unipolar depressed patients showed

smaller P2 slopes, but only when medication was not controlled. Implications of these results for further research on augmenting/reducing and affective disorders and their relationship to SS are discussed.

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Introduction

A number of findings suggest a positive relation between sensation seeking (SS) and the augmenting disposition of the evoked potential (EP) of the EEG [1]. When stimulus intensity increases, sensation seekers respond with a marked increase in the P1/N1 or N1/P2 component of the EP. This change is also referred to as augmenting disposition or 'high-level intensity dependence'. Since Zuckerman's [1] review, this finding has been replicated several times [2, 3]. In a recent study, we could also confirm the correlation between SS and augmenting [4]. However, some of the inconsistencies in the earlier findings still require clarification. Methodological problems concern the lack of comparability of various EP parameters that were used [5].

There are indications that particularities of the intensity dependence also appear in certain psychological disorders, for instance in alcoholism and affective disorders [6, 7]. Following the basic work by Buchsbaum et al. [6], several studies found that patients with bipolar affective dis-

orders (BAD) show a pronounced augmenting disposition in comparison to healthy controls. Another finding is that responders to antidepressant or prophylactic medication are augmenters, whereas nonresponders tend to be reducers [7, 8]. With regard to unipolar depression (UPD), Buchsbaum's findings suggest that these patients exhibit a significantly lower intensity dependence than did BAD patients and healthy controls, thereby showing themselves to be reducers [9].

Thus far, several findings suggest that sensation seekers (SS) and BAD share psychophysiological characteristics, and SS and UPD patients might differ with regard to augmenting/reducing. This relationship could turn out to be mutually indicative of the SS trait and affective disorders if further findings support the assumption that a high level of intensity dependence in EP can be taken as an indicator of low central serotonergic neurotransmission, particularly in the primary auditory cortex [7]. Peculiarities of central serotonergic neurotransmission are assumed to be of importance for both SS and affective disorders [7, 10].

In addition, the question arises as to whether BAD and SS are also associated behaviorally or psychometrically, and in particular whether SS might be a vulnerability factor for the development of a bipolar disorder. The psychophysiological findings described thus far support this assumption and correspond well with initial psychometric findings on the relation between SS and affective disorders. Using the SS Scale Form V (SSS-V) [11], Zuckerman and Neeb [12] were able to distinguish between controls and patients with a reported history of bipolar depression. Cronin and Zuckerman [13] showed that inpatients with bipolar depression had significantly higher SS scores (sub-scales disinhibition and boredom susceptibility) than controls, though half of the patients were depressed at the time of the study.

Little is known about the effects of antidepressant and prophylactic medication with serotonergic relevance on the augmenting/reducing characteristic of BAD and UPD. The study by Buchsbaum et al. [6] revealed a significant decrease in the intensity dependence of BAD under lithium ('flattening effect'). Some studies found the same effect on the intensity dependence for different patient groups when tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRI) were used [14, 15, summarized in 7]. Overall, it has not been clarified yet whether the flattening effect is stable or only transient during the first time of medication, since appropriate studies with retests over a respective period are not available. Thus, the question arises whether the stronger intensity dependence of BAD patients compared to healthy con-

trols under antidepressant or prophylactic medication really changes in favor of a flattening effect, and whether the intensity dependence of both groups is similar under these conditions.

The study by Buchsbaum et al. [6] does not give evidence of changes on the intensity dependence of unipolar depressed patients while treated with lithium. So far, studies on the effect of antidepressant medication with UPD patients have been mainly restricted to clinical responsiveness studies [8, 16].

All in all, the actual findings concerning the intensity dependence of antidepressant or prophylactically medicated BAD and UPD patients in comparison to healthy controls do not offer sufficient information yet. The present study investigates the intensity dependence of SS in healthy individuals. In addition, the aim of the study was to investigate differences in the intensity dependence between both affective-disordered groups (BAD, UPD) and in comparison to healthy individuals. At the psychometric level, it is attempted to replicate the findings of higher SS scores for BAD and lower SS scores for UPD when compared to controls.

Methods

Participants

Inpatients with diagnoses of affective disorders were recruited from the State Mental Hospital Arnsdorf, Germany. Patients' participation did not interfere with their therapeutic treatment. Hospital staff and interested volunteers were recruited as controls ($n = 24$; age: mean = 47, SD = 11; 16 females) and screened for current or past history of psychiatric illness and substance abuse. Written informed consent was obtained from all participants.

A structured clinical interview [17] was taken by a trained graduate student, and consensus diagnosis was established together with an experienced psychiatrist (H.H.). Patients with ambiguous diagnoses ($n = 17$) were not included in the analysis. Furthermore, patients were excluded if they had a past history of organic brain disorder or substance abuse ($n = 5$). We obtained EEG retest data from 27 of the final sample of 42 patients.

According to the International Classification of Diseases [18], 21 patients of the final sample were diagnosed as suffering from UPD (age: mean = 47, SD = 11; 12 females); of these, 20 patients were classified as having a depressive episode (F32), and 1 patient as having a recurrent depressive disorder (F33). The second clinical group consisted of 21 patients with a diagnosis of BAD, who were in different episodes at the recording time (BAD; F31; age: mean = 43, SD = 12; 8 females). Seven of the 21 BAD patients had psychotic symptoms, whereas none of the UPD patients had psychotic symptoms during the time period of the study. There were no significant differences in age and gender distributions between BAD, UPD and the control group.

Medication status for all patients was monitored over the interval of the study. The majority of the UPD patients received antidepressant medication (13 tricyclic antidepressants, 3 SSRI). Two patients

received prophylactic medication (lithium), 2 neuroleptic medication. One patient received anxiolytic medication only, whereas 12 patients received anxiolytic comedication (benzodiazepines). One UPD patient was unmedicated.

The BAD patients received prophylactic (9 lithium, 5 carbamazepine) or antidepressant medication (3 tricyclic antidepressants, 3 SSRI). Two of them received both antidepressant and prophylactic medication and 12 anxiolytic or neuroleptic medication as comedication.

Participants were requested to fill in self-report personality questionnaires. Special interest was focused on the SSS-V [11] which consists of the four subscales thrill and adventure seeking (TAS), disinhibition (DIS), experience seeking (ES), and boredom susceptibility (BS).

Data Collection and Quantification

In order to check the stability of the EEG measurements, a retest was scheduled 2–4 weeks after the initial recording session. Separate diagnostic sessions, including the assessment of personality dimensions and clinical state, were scheduled within a few days of the EEG recording sessions. All recordings were scheduled at fixed times and intervals in the morning.

After audiometric screening of participants, EEG data were recorded from 19 standard scalp sites (10–20 system) using a forehead ground and linked earlobes as reference, while participants sat in a comfortable arm chair with their eyes open. EEG data were band pass filtered (0.1–30 Hz) and continuously digitized at 256 samples/s. To facilitate artifact rejection, the vertical electro-oculogram was recorded from additional electrodes placed above and below the right eye. After a recording of 8 min of resting EEG, the acoustic augmenting/reducing paradigm [19] was administered (13 min time duration). For a more accurate calculation of the intensity dependence, six different intensity levels were used (59, 71, 79, 88, 92, 96 dB SPL). Stimuli were presented binaurally via headphones in a pseudorandomized order. Each 1,000-Hz stimulus lasted 30 ms (10 ms each of rise and fall time), the interstimulus interval varied between 1,600 and 2,100 ms.

EEG data were epoched off-line from 100 ms before to 400 ms after stimulus onset. Following baseline correction of the prestimulus interval to 0 μ V, epochs with artifacts were excluded automatically if the EEG signal exceeded ± 50 μ V prestimulus or ± 65 μ V poststimulus. Long-latency acoustic EP (AEP) were obtained by averaging the remaining epochs for each stimulus intensity separately (minimum sweep number 20 epochs). Peak amplitudes were determined for the P1 as the most positive peak within the period of 25–80 ms, for the N1 as the most negative peak within 55–135 ms, and for P2 as the most positive peak within 110–260 ms. Additional peak-to-peak values were calculated for P1/N1 and N1/P2.

Statistical Analysis

Similar to previous studies, the slopes of the linear regression line (least-squares technique) for the individual P1, N1, P2, P1/N1 and N1/P2 amplitudes were calculated across the six stimulus intensities. However, because the use of regression slopes for the amplitude stimulus intensity function (ASF) has been criticized for their low coefficients of determination in individual data [20], a nonlinear computation was also performed [2]. The medians of all possible slopes between each two amplitudes for the six stimulus intensities (median slopes) were calculated. All parameters were calculated for C3, Cz and C4 scalp locations [1].

Table 1. Retest reliabilities of the augmenting/reducing parameters in controls and patients for ASF slopes and median slopes at three central scalp sites, separately

Parameters	ASF slopes			Median slopes		
	C3	Cz	C4	C3	Cz	C4
<i>Controls (n = 24)</i>						
P1	0.18	0.02	0.14	0.04	−0.08	−0.08
N1	0.38	0.07	0.06	0.08	−0.27	−0.01
P2	<i>0.61</i>	<i>0.77</i>	<i>0.67</i>	<i>0.47</i>	<i>0.65</i>	<i>0.50</i>
P1/N1	0.19	0.30	0.31	0.27	0.17	<i>0.50</i>
N1/P2	<i>0.77</i>	<i>0.71</i>	<i>0.59</i>	<i>0.79</i>	<i>0.80</i>	<i>0.64</i>
<i>Patients (n = 28)</i>						
P1	0.20	0.25	<i>0.43</i>	0.24	0.34	0.29
N1	<i>0.53</i>	<i>0.58</i>	0.21	<i>0.53</i>	<i>0.41</i>	0.28
P2	<i>0.41</i>	<i>0.61</i>	<i>0.47</i>	0.23	<i>0.42</i>	0.17
P1/N1	0.22	0.34	0.17	<i>0.41</i>	<i>0.43</i>	<i>0.43</i>
N1/P2	0.17	0.38	0.18	0.03	0.22	0.11

Pearson correlations >0.40 are in italics.

Preliminary analyses addressed the retest reliability of the different computations of augmenting/reducing to evaluate effects of electrode site, AEP components, and the method of parametrization for patients and healthy controls separately. A second recording session was available from 14 BAD, 14 UPD and 24 control participants. Additional correlational analyses focused on the relationship of the augmenting/reducing tendency with the SS trait for healthy participants only.

To examine the group differences in augmenting/reducing, data from the first recording session were submitted to a 2 $\times$ 3 repeated-measures analysis of variance (ANOVA), including position (C3, Cz, C4) and group. Since we assumed different effects comparing UPD, BAD and controls, the group differences were tested separately. Additional analyses included session as a repeated-measures factor for those participants where a retest was available. Comparisons for all pairs of groups (UPD vs. controls, BAD vs. controls, UPD vs. BAD) are reported for ASF slopes only, because analyses for median slopes revealed similar results.

To be sure that no group effects are interpreted that may be related to the effects of antidepressants, medication effects were controlled by means of covariates. The applied medications were coded as yes (1) or no (0). However, the influence of the covariates is not independent of the frequency of the medication applied. The total number of participants receiving partially similar medication is different for the UPD and BAD groups. Therefore, it is important to control for medication, in order to control for effects due to different frequencies of medication in the groups. The covariates represent the direct influence of a medication on augmenting/reducing together with the frequency of the respective medication and therefore have to be interpreted with caution.

Differences in SS between UPD, BAD and controls were calculated by means of t tests, for the SSS-V total scale and the four subscales.

Fig. 1. Grand average AEP for UPD patients ($n = 21$), patients with BAD ($n = 21$), and healthy controls ($n = 24$) for six different stimulus intensities at recording site Cz. The AEP components P1, N1 and P2 are indicated for BAD patients at 59 dB SPL.

Results

Figure 1 shows the AEP for the scalp site Cz for UPD patients (left column), healthy controls (middle column) and BAD patients (right column). Averages are separately displayed for each stimulus intensity level from 59 dB (bottom) up to 96 dB (top). Independent of group, the P1, N1, and P2 components of the AEP were apparent at each level of stimulus intensity. Overall, the augmenting tendency was visible in each group, i.e. a pronounced amplitude increase with increasing stimulus intensity.

Reliability of the AEP Parameters (Intensity Dependence)

The following test-retest reliabilities (temporal stability) were computed for the control group ($n = 24$) and the patient group ($n = 28$) separately. The retest reliabilities for the parameters differed considerably in both patients and controls (table 1).

For controls, satisfactory retest reliabilities were achieved for the ASF slopes for P2 and N1/P2, irrespective of the electrode site. The slopes of the linear regression line for the P1, N1, and P1/N1 amplitudes were not

Table 2. Pearson correlations of the ASF slopes with the SSS-V total score in controls (n = 24)

Parameters	First recording session			Second recording session			Aggregated across sessions		
	C3	Cz	C4	C3	Cz	C4	C3	Cz	C4
P1	0.34	0.27	0.29	0.11	0.15	0.26	0.29	0.30	0.37*
N1	0.30	0.30	0.37*	0.12	0.04	0.22	0.24	0.20	0.40*
P2	0.29	0.37*	0.35*	0.45*	0.34*	0.41*	0.41*	0.38*	0.42*
P1/N1	0.01	-0.01	-0.10	-0.04	0.08	-0.01	-0.02	0.04	-0.06
N1/P2	0.07	0.16	0.00	0.27	0.23	0.16	0.18	0.21	0.09

* $p < 0.05$, one tailed.

reliable (table 1). When compared to the ASF slopes, the reliabilities of the median slopes were not consistently different. For patients, the pattern of reliabilities was less consistent in comparison to healthy participants. Whereas the N1/P2 slope was not reliable for patients, acceptable values were obtained for the N1 slopes on C3 and Cz, and for the P2 slopes on all sites. The pattern of retest reliabilities was different for ASF slopes and median slopes. However, across both groups, method of parameterization did not affect reliabilities consistently.

SS and Intensity Dependence of the AEP for Healthy Participants

On both measurement sessions, the most obvious relationships with the SSS-V total score were found for the P2 slopes, the parameters with the highest reliabilities. This result remained stable when measurements were pooled across sessions. The SSS-V total score correlated with the P2 slopes, which were reliable, but it correlated only inconsistently with the P1 and N1 slopes (table 2). P1 and N1 slopes were not reliable in healthy participants. For the N1/P2, only a correlation with the SSS-V subscale TAS was found ($r = 0.36$, $p < 0.05$, one tailed) on C3 in the second recording session. Pooled across sessions, the slopes of the P1 and N1 on C4 showed significant relations with the SSS-V total score. Since the correlations of P1 and N1 slopes with SSS-V scores indicated some validity of these parameters, we included them into further analyses. The expected positive correlation between SS and the intensity dependence of the AEP was confirmed again by the present results.

Clinical Groups and Intensity Dependence

For the first session, a significant main effect was found for P1/N1 slopes, when controlled for medication,

confirming the assumption of BAD patients having larger slopes, when compared to controls, $F(1, 37) = 5.91$, $p < 0.05$. The same effect remained significant without BAD patients medicated with neuroleptics, when controlled for medication, $F(1, 30) = 8.50$, $p < 0.01$. Similarly, a tendency was found for the P1 slopes when controlled for medication, $F(1, 37) = 3.88$, $p = 0.056$.

For UPD patients compared to healthy controls, a significant group effect was found between the P2 slopes of UPD patients and controls, $F(1, 43) = 5.91$, $p < 0.05$, with controls having a larger slope. When controlled for medication, this effect disappeared, $F(1, 38) = 1.34$, n.s. Calculated without the UPD patients treated with neuroleptics, the effect was significant, $F(1, 41) = 5.69$, $p < 0.05$, but also disappeared when controlled for medication, $F(1, 37) = 0.74$, n.s. A tendency was found for groups when N1/P2 slopes of UPD patients were compared to controls. Again, UPD patients had smaller slopes when compared to healthy controls, $F(1, 43) = 3.68$, $p = 0.062$. This tendency was not significant when controlled for medication, $F(1, 38) = 0.42$, n.s. Without the UPD patients treated with neuroleptics, there was a similar tendency for the N1/P2 slopes, $F(1, 41) = 3.18$, $p = 0.082$, which disappeared when controlled for medication, $F(1, 37) = 0.00$, n.s. The effects of the covariates for tricyclic antidepressive drugs and SSRI were mainly responsible for this shift.

For the first recording session, none of the comparisons between BAD patients and UPD patients reached significance, even when controlled for medication.

In the ANOVAs for patients with two AEP recording sessions (15 with UPD, 12 with BAD), there was only a main effect for UPD patients having smaller N1/P2 slopes than controls, $F(1, 37) = 5.01$, $p < 0.05$. A similar main effect for the P2 slopes occurred, $F(1, 37) = 4.90$, $p <$

0.05. No main effect for recording session nor any group $\times$ session interaction was found. Despite of changes in the therapeutic medication of several patients, the effects remained stable across the recording sessions.

Clinical Groups and SS

As expected, BAD patients had significantly higher SSS-V total scores than UPD patients, $t = -2.29$, $p < 0.05$ (this and the following significances reflect one-tailed tests). BAD patients scored significantly higher than UPD patients on the subscales ES, $t = -2.30$, $p < 0.05$, and DIS, $t = -2.89$, $p < 0.01$. In addition, BAD patients had greater SSS-V total scores than controls, $t = -1.80$, $p < 0.05$, and scored higher on the DIS subscale, $t = -2.59$, $p < 0.05$. For the differences between UPD patients and controls, there was – again in accordance with the assumptions – a tendency for ES, $t = 1.43$, $p = 0.08$, with UPD patients scoring lower.

The confirmed relationship of SS with ASF slopes in healthy participants, as well as significant group differences in SS, may have modulated group differences for ASF slopes. We therefore reanalyzed our data. The corresponding position $\times$ group $\times$ SS ANOVA was based on a median split of the SSS-V total score of group-specific medians. These analyses did not reveal any significant interaction involving SS for either the first or the second recording session.

Discussion

At the psychophysiological level, we were again able to replicate the positive correlation between augmenting and SS [4, 10]. These findings are especially informative since the study yields data on the reliabilities of the parameters used. The correlations with SS were again confirmed for the P2 slopes in healthy participants. This result may be due to remarkable reliabilities of this parameter. However, the N1/P2 slopes, which showed comparable reliabilities, only correlated with the TAS subscale. For healthy participants, inconsistent correlations of the N1 and P2 slopes with SS emerged. This goes parallel with low reliabilities of the N1 and P2 slopes in this study.

In addition, we found the expected steeper ASF slopes for BAD patients, i.e. augmenting, when controlled for medication. Without controlling for medication, no effects reached significance for ASF slopes. Thus, it is possible that the medication of the BAD patients flattens the slopes, and therefore eliminates the differences between BAD patients and controls [6, 7]. When calculated with-

out patients with neuroleptic medication, the results remained similar, so that the flattening effect may not be due to neuroleptic medication. However, further studies with more homogeneously medicated patients are necessary in order to assess the influence of different types of medication on individual differences in ASF slopes.

Our results indicate that UPD patients have smaller ASF slopes than controls, but this does not hold when controlled for medication. This means that the medication of the UPD patients could be responsible for their smaller ASF slopes. On the other hand, it might be possible that UPD patients would still have smaller slopes without medication, as reported by Buchsbaum et al. [6]. The analyses across recording sessions point into this direction because UPD patients have smaller ASF slopes than controls even when medication changes. The changes in medication in UPD patients across recording sessions had no effect on the ASF slopes since there was no significant change in the slopes across sessions. This, furthermore, indicates that the smaller slope of the UPD patients cannot be totally attributed to the effects of medication.

Overall, the results must be regarded with caution, because it was only possible to control medication effects on the augmenting/reducing disposition by means of statistical analysis. Even if equal group size for every type of medication and comedication could be obtained, there will always be confounding influences left (e.g. type of disease and medication), which cannot be completely controlled for. However, these limitations are typically given in naturalistic settings, since it is neither possible nor recommended to leave patients unmedicated.

At the psychometric level, the differences in SSS-V scores confirmed our assumption of BAD patients having higher scores than UPD patients and controls. This is a further replication of the results of Zuckerman and Neeb [12], Cronin and Zuckerman [13] and Carton et al. [21]. Some of the results of Carton et al. [21] could only be replicated with reservations, because there was only a tendency for the difference between UPD patients and controls. However, SS did not modulate group differences in ASF slopes, as revealed by reanalyses. The group-specific differences in SS did not account for additional variance in ASF slopes. This finding suggests that group characteristics and SS are highly dependent and that they account for a large amount of common variance in ASF slopes.

Overall, some psychophysiological and psychometric differences between BAD and UPD patients and controls could be demonstrated, which were in line with the few findings on these relations when controlled for medica-

tion. In addition, when effects of prophylactic or antidepressant medication are not statistically controlled, the relations are also compatible with the reported characteristics of the intensity dependence of medicated BAD, i.e. the flattening effect of the slopes when medicated. However, due to the naturalistic setting of this study, these results must be taken with caution and can only be seen as an indication. Future studies, especially with more restricted medication or with pre/postdesigns, are needed for further clarification of the characteristic intensity dependence of unmedicated BAD and UPD patients and the effects of antidepressant and prophylactic medication

on these groups. However, this is not easily accomplished. Besides, the prognostic value of SS and augmenting/reducing in the clinical context might be more appropriately assessed in a naturalistic setting.

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