Semantic Processing of Emotional Words in Depression and Schizophrenia

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Abstract

Major depressive disorder is associated with dysfunction in brain regions involved in language and emotion processing. Despite evidence of emotion processing biases in depression, neurophysiological evidence of language dysfunction for emotional words in depression has been inconsistent. This series of three studies evaluated whether depressed individuals exhibited abnormal semantic processing of emotionally valenced words. During the passive viewing of sentences with mood congruent and incongruent sentence endings, the N400 component of the event-related brain potential was measured in patients with depression, dysthymia, or schizophrenia and in healthy controls. In each study, results revealed normal semantic processing in depression. That is, N400 was similar for both mood-incongruent (positive and neutral) endings and mood-congruent (negative) endings. In contrast, the small sample of individuals with schizophrenia exhibited a significantly exaggerated N400 for negative word endings compared to the depressed and healthy control groups. These data suggest anomalies in semantic network interactions with emotion processing in schizophrenia.

Key Words: depression, schizophrenia, semantic processing, N400, emotional words, biases, ERP
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Introduction

Major depressive disorder (MDD) is a prevalent and disabling psychiatric illness that has been hypothesized to be associated with a general dysfunction in the left frontal lobe (e.g., Davidson et al., 2002; Heller and Nitschke, 1997). Bilateral dysfunction in the dorsolateral prefrontal cortex (DLPFC) has also been proposed (Johnstone et al., 2007; Levin et al., 2007; Mayberg, 2006). Although these brain regions are involved in language processing, individuals with depression do not have consistent language deficits (for a review see Klumpp and Deldin, this issue) and do not show semantic processing difficulties with neutral words manifested in the N400 component of the event-related brain potential (ERP) (Deldin et al., 2006; Iakimova et al., 2009), an index of semantic processing (Kutas and Hillyard, 1980a, b). The failure to find consistent language dysfunction suggests that the purported left frontal or bilateral DLPFC deficits may be task-specific and would necessitate a revision of models away from particular cognitive deficits towards more complex neural network models that account for interactions among brain regions (e.g., cortical-subcortical). Support for this hypothesis and the potential for individual differences in neural function is supported by a review of brain functioning in depression (Fitzgerald et al., 2008), which noted inconsistent results across studies. These investigators propose that the pathophysiology of depression is likely complex and not amenable to simple models. For example, it may be the case that language dysfunction in depression is more likely to manifest when emotional stimuli are being presented, as the potential for dysfunctional neural interactions that involve emotion processing is increased. Behavioral evidence in support of this includes studies that show that individuals diagnosed with MDD are more likely to have cognitive biases towards negative stimuli (Caseras et al., 2007; Gotlib et al., 2004; Mathews et al., 1996) or away from positive stimuli (e.g., Deldin et al., 2001; Joormann and Gotlib, 2006; Surguladze et al., 2004; Yoon et al., 2009) than general non-emotional language processing deficits.
Regarding semantic processing, behavioral studies of depression show equivocal evidence of dysfunction for emotional words. For example, some provide evidence of biased responses to negative words (enhanced response latency for negative words; Weisbrod et al., 1999), partial evidence of such bias (Atchley et al., 2003), and lack of evidence of semantic dysfunction (Dannlowski et al., 2006). Other behavioral studies show normal semantic processing for neutral words in MDD (Besche-Richard et al., 2002; Georgieff et al., 1998). Thus, relatively few behavioral studies have investigated semantic processing of emotional words, and the results of this limited literature are inconsistent.

Neurophysiological studies examining emotional language processing are also equivocal, and results depend upon the specific process examined. For example, some ERP components detect enhanced processing of emotional words in depression (e.g., P300, P2, N2, PINV; Nikendei et al., 2005; Serfaty et al., 2002; Shimizu et al., 2006), whereas N100 and CNV (Nikendei et al., 2005; Serfaty et al., 2002) indicate normal language processing in depression. However, semantic processing of emotional words as indexed by N400 has not been evaluated in MDD.

In the present series of three studies, MDD and healthy control participants were presented sentences with mood congruent and incongruent emotional endings in order to assess whether individuals diagnosed with mood disorders have normal emotional semantic processing. Participants passively viewed the sentences while their ERPs were recorded. If emotional semantic processing errors occurs in depression, participants will display increased N400 for emotionally incongruent (i.e., positive word) endings compared to healthy controls. Alternatively, if language processing, regardless of emotional valence is normal in depression, no differences between the depressed and healthy control groups will be found.

One of the three studies included dysthymic participants in order to determine if semantic processing deficits extended to a second mood disorder group. Another of the studies included individuals diagnosed with schizophrenia, as language disturbance (e.g., abnormal
associations) is a hallmark of this illness and should be associated with N400 abnormalities (Adams et al., 1993; Hokama et al., 2003; Kiang et al., 2007; Kostova et al., 2003, 2005; Ohta et al., 1999; Sitnikova et al., 2002; Strandburg et al., 1997). Moreover, there is evidence of abnormal emotion processing in schizophrenia (An et al., 2003; Bell et al., 1997; Berenbaum et al., 2008; Dougherty et al., 1974; Kring and Moran, 2008; Suslow et al., 2003). Hence, schizophrenia is a relevant comparison group for MDD and may highlight the sensitivity of the task in detecting N400 anomalies.

Methods

Participants

Individuals diagnosed with major depressive disorder (MDD) were recruited from an acute inpatient psychiatry unit in a moderate-size, mid-western city (Studies 1 and 2) and from a large eastern city (Study 3). Individuals diagnosed with schizophrenia (SCZ) were recruited from an inpatient unit (Study 2). Individuals with dysthymia and non-patient comparison participants were recruited from both communities via newspaper advertisements and paid $5-$10/per hour for participation. Inpatient MDD and SCZ participants were not paid for participation. For all studies, left-handed participants were excluded. Participants were diagnosed with current MDD (Studies 1-3), dysthymic disorder (Study 3), or schizophrenia (Study 2) using the Structured Clinical Interview for the DSM-III-R (Study 1) or DSM-IV (Studies 2 and 3) (SCID; First et al., 1996; Spitzer et al., 1990). To ensure diagnostic consensus, interviews for Studies 1 and 2 were completed by advanced doctoral students in clinical psychology closely supervised by a clinical psychologist (GAM). In Study 3, a clinical psychologist (PJD) and advanced graduate students conducted the clinical interviews. Participants were included in the study only if a consensus diagnosis was reached by trained supervisees of PJD in a review of taped interviews.

Physiological Recording
The electroencephalogram (EEG) was recorded from nine International 10-20 System sites (Jasper, 1958): Fz, Cz, Pz, F3, C3, P3, F4, C4, and P4, referenced to the left mastoid (Studies 1 and 2) or left earlobe (Study 3). EEG activity recorded from the right mastoid or right earlobe was subsequently used to compute an average mastoid reference ([A1+A2]/2; Miller et al., 1991). In Studies 1 and 2, Beckman miniature Ag-AgCl electrodes were used for the EEG recording. In Study 3, EEG was collected using a tin-electrode cap (Electro-Cap International, Inc., Eaton, OH). For all three studies, two orthogonal channels of the electro-oculogram (EOG) recorded vertical and horizontal eye movements using electrodes placed near the outer canthi and at left supra- and suborbital sites. Impedances were kept below 10 kΩ.

EEG was amplified using a Grass Model 12 (Studies 1 and 2) or an S. A. Instruments Custom Bioamp polygraph (Study 3) with a .01-30 Hz analog bandpass filter. Data were sampled at 125 Hz (Studies 1 and 2) or 512 Hz (Study 3) for 1400 ms beginning 200 ms prior to the onset of the last word in each sentence.

Procedure

After an initial interview that screened prospective participants for handedness, head injury, seizures, normal or corrected-to-normal vision, major medical illness, and developmental disabilities, eligible participants were given an individual lab tour and provided written consent prior to the SCID diagnostic interview.

Participants returned within 30 days for the electrophysiological portion of the study. On average, the length of time between the diagnostic interview and EEG study was 2 weeks. Following electrode application, participants were instructed to relax and focus on the screen in front of them. Consistent with other depression and schizophrenia studies (e.g., Adams et al., 1993; Deldin et al., 2006; Kuperberg et al., 2006), sentences were presented one word at a time. Thus, participants were told that the briefly presented words formed sentences. They were asked to read the sentences silently and told that they would be asked questions about the
sentences at the end of the session. Subjects were then presented with a block of 30 sentences; each sentence stem was repeated three times, once each with the terminal word being a positive, neutral, or negative word (e.g., Today I am feeling (happy, okay, or sad)). The order of sentence terminal word ending (i.e., negative, positive, neutral) was random within the constraints that no more than four of the same type occurred consecutively. Additionally, sentences were counterbalanced such that the first presentation, used for N400 analyses, varied in emotional ending. Sentences were 6-8 words in length. The inter-stimulus interval was 400 ms: each word appeared on the screen for 200 ms, with 200 ms of blank screen between each word. An inter-trial interval of 2600 ms occurred between the offset of the last word of a sentence, indicated by a period, and the onset of the next sentence.

**Stimuli.** First, a list of emotional adjectives was compiled from several studies (Anderson, 1968; Bellezza et al., 1986) and was supplemented by the experimenters. Students then rated each word on five-point Likert scale for happy/sad, arousing/calm, and dominant/passive. Valence ratings for the chosen types of words (positive, neutral, and negative) were found to be significantly different, $F(4,85)=346.58, p<.001$. Words were also classified as either high or low arousal. Emotional words (positive and negative) had reliably high and similar arousal ratings relative to neutral words, $F(4,85)=152.28, p<.001$. In addition, average length, $F(4,85)=1.66, p<.17$, and frequency, $F(4,85)=.14, p<.97$, of the terminal words were comparable across stimulus conditions. A total of 90 emotion sentences were then developed using a base sentence and various terminal words chosen using the ratings discussed above. The sentences were the same across the three studies.

**Data Reduction and Analyses**

Eye movement correction procedures were performed (Gratton et al., 1983; Miller et al., 1988, for Studies 1 and 2; James Long Co., unpublished, for Study 3). Sentence type was categorized according to the valence of the word (positive, neutral, or negative) that ended the
sentence. EEG was manually inspected for the presence of residual eye movements or EMG artifact, and contaminated epochs were removed from the analysis. Within-subject average ERP waveforms were computed for each site and valence type. N400 scores were computed as the average voltage between 300 and 600 ms deviated from a 200 ms pre-stimulus baseline.

Data analysis. For each study, N400 scores collapsed across the midline (Fz, Cz, Pz) were submitted to a Group [MDD, nondepressed (Studies 1, 2 and 3); MDD, SCZ, nondepressed controls (Study 2); or MDD, dysthymia, nondepressed (Study 3)] x Valence (positive, neutral, negative) repeated-measures analysis of variance (ANOVA). ANOVAs were supplemented with simple-effects ANOVAs and Bonferroni tests as needed to interpret effects significant at the two-tailed, 0.05 level. Reported probability values reflect the Huynh and Feldt (1976) degrees of freedom correction. Finally, consistency of the three individual studies were then confirmed using the Stouffer meta-analytic procedure (Bush et al., 1954) to determine whether each group exhibited statistically significant N400 scores (area measurement) for positive, neutral, or negative words. In this procedure (Bush et al., 1954), p values for the studies were converted to z scores, which were summed and divided by the square root of the number of studies.

Results

Participant Characteristics

Demographics. Across studies demographic characteristics were similar among MDD, dysthymia, and healthy control groups (see Table 1). The schizophrenia group had proportionally more males and was less educated than the other groups. These characteristics
are consistent with evidence that males have a higher lifetime risk of developing schizophrenia (Tandon et al., 2008) and are less likely to complete high school (e.g., Carr et al., 2004).

Within-Study Results for Positive, Neutral, and Negative Words

Figure 1 present ERP grand averages for each study, and Tables 2 and 3 provide ANOVA results. Across all three studies, there was no difference in N400 amplitude between the healthy controls and the depressed groups (both major depression and dysthymia). This result was confirmed with the meta-analytical procedure (see Table 2). The only significant result concerned the SCZ group (Study 2). Follow-up 3 (Group: MDD, SCZ, control) x 3 (Valence: negative, positive, neutral) simple-effects ANOVAs were conducted for each word type. Differences were found for negative ($F(2, 32)= 4.28, p<.02$) but not positive ($F(2, 32)= 1.51, p=.24$) or neutral ($F(2, 32)= 2.34, p=.11$) words. Post-hoc Bonferroni comparisons for negative word endings revealed larger N400 in schizophrenia patients compared to major depression ($p<.05$) and healthy control ($p<.03$) groups. There was no difference between the MDD and healthy controls ($p=.99$).

Discussion

N400 results suggest normal language function during emotion processing in depression. Thus, N400 as a measure of semantic integration (Brown and Hagoort, 1993) is not impaired for emotionally-valenced words. These results point to models of depression that
emphasize diverse functional disruptions in neural networks (e.g., Fitzgerald et al., 2008; Mathews et al., 2008) rather than discrete cognitive deficits. For example, cortical-subcortical responses to cognitive and emotional tasks have been shown to differ among depressed individuals (Siegle et al., 2007). Additionally, brain imaging studies do not consistently show lateralized effects in depression or reliable neural patterns (e.g., Fitzgerald et al., 2008). Taken together, accumulating data indicate that abnormal function among brain regions, possibly to varying degrees (e.g., strength of cortical-subcortical interactions, neural compensatory processes) may be a factor in individual differences in depression rather than specific deficits (e.g., hypoactive left frontal lobe). Thus, although particular neural networks are implicated in depression, the varying ability of a given task to tap into abnormal function and individual differences in neural network function may help explain mixed results in language processing for emotional words.

Of note, schizophrenia patients exhibited a larger N400 (more negative response) to negative words than the depressed or healthy controls, and there was no difference between these groups in response to positive or neutral sentence endings. This provocative finding suggests a violation of expectancy only for the negative words, indicating a greater surprise to negative sentence endings. This finding is consistent with studies that show impaired processing of emotional stimuli in schizophrenia (e.g., for review see Gold et al., 2009; Rockstroh et al., 2006). Interestingly, the results in this study were specific to negative valence, whereas other studies have found differential processing for both positive and negative valenced stimuli compared to neutral stimuli. While the specific finding argues against a general deficit, we cannot rule out the possibility that factors such as reading ability and/or working memory deficits may have impacted results.

Given the small sample size of the schizophrenia group, results are in need of replication. Nevertheless, results suggest that schizophrenia patients have a relatively large violation of expectancy to negative sentence endings than do depressed patients and healthy
controls. This finding may help elucidate cognition-emotion interactions in schizophrenia. For example, operation of the semantic network structure in schizophrenia is not entirely clear though it has been recently proposed that problems in schizophrenia involve both an initial overly activated semantic network and later inhibition difficulties (e.g., failure to rely on context) (Niznikiewicz et al., in press). The present significant results were restricted to negative word endings to ambiguous sentences. Therefore, it may be the case that these stimuli reflect problems integrating negatively-valenced stimuli within a vague context though further study is needed to better understand the extent to which results reflect early or later semantic network abnormalities.

Limitations of this study include the broad scoring window used to measure N400 (i.e., 300 ms to 600 ms) to ensure N400 was captured in patient groups. It is possible that results for some participants may include some later positivity. Additionally, the sample sizes in each study, particularly schizophrenia patients, were small, which may have reduced our ability to find within group differences based on the meta-analytic calculations. All the schizophrenia patients were on medications, and we could not control for medication in that group. Also, although the word endings were in the first person and intended to be self-referential, the same words were used in each study. Therefore, it is possible that individual differences for self-referent emotional words reduced sensitivity to detect N400 effects for mood-incongruent words.

Despite limitations, this study provides further evidence for normal N400 in MDD and dysthymic participants. This study expands on N400 studies of neutral stimuli by suggesting N400 to emotional stimuli is also normal in MDD and dysthymia. Finally, this study demonstrates that schizophrenia patients exhibit abnormal information processing as indexed by enhanced N400 to negative stimuli thus providing further evidence of the specificity of cognitive biases in mood disordered participants and disruption of emotional and language processing in schizophrenia.
References


Bell, M., Bryson, G., Lysaker, P., 1997. Positive and negative affect recognition in schizophrenia: a comparison with substance abuse and normal control subjects. Psychiatry Res. 73, 73-82.


Joormann, J., Gotlib, I.H., 2006. Is this happiness I see? Biases in the identification of
emotional facial expressions in depression and social phobia. J. Abnorm. Psychol. 115, 705-714.


### Table 1

*Means and Standard Deviations in Parentheses for Participant Characteristics*

<table>
<thead>
<tr>
<th>Study 1</th>
<th>N</th>
<th>Medication</th>
<th>Age</th>
<th>Education</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>MDD</td>
<td>17</td>
<td>70.6%</td>
<td>35.1 (9.7)</td>
<td>14.12 (2.0)</td>
<td>57.1%</td>
</tr>
<tr>
<td>Controls</td>
<td>12</td>
<td>0%</td>
<td>31.7 (12.9)</td>
<td>14.75(1.5)</td>
<td>36.4%</td>
</tr>
</tbody>
</table>

| Study 2          |               |          |       |           |        |
| Schizophrenia    | 7             | 100%     | 33.2 (9.0) | 11.00 (.71)* | 14.3%* |
| MDD              | 12            | 100%     | 31.0 (9.2) | 14.29 (2.0)  | 75.0%  |
| Controls         | 14            | 0%       | 44.7 (15.5) | 15.75 (1.5)  | 57.1%  |

| Study 3          |               |          |       |           |        |
| MDD              | 18            | 27.8%    | 40.3 (13.1) | 14.50 (2.7)  | 50.0%  |
| Dysthymia        | 14            | 14.3%    | 42.7 (13.4) | 15.38 (2.1)  | 71.4%  |
| Controls         | 19            | 0%       | 36.8 (14.6) | 16.07 (2.0)  | 52.6%  |

*Significantly different from major depressive disorder, dysthymia, and healthy control groups (p<.05).*
Table 2. *ANOVA Results Within Study and Meta-Analysis Results*

a. Group main effects

<table>
<thead>
<tr>
<th>Study</th>
<th>Groups</th>
<th>d.f.</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Control, MDD</td>
<td>1, 27</td>
<td>.23</td>
<td>.64</td>
</tr>
<tr>
<td>2</td>
<td>Control, MDD</td>
<td>1, 24</td>
<td>1.97</td>
<td>.17</td>
</tr>
<tr>
<td>2</td>
<td>Control, MDD, Schizophrenia</td>
<td>2, 30</td>
<td>1.90</td>
<td>.17</td>
</tr>
<tr>
<td>3</td>
<td>Control, MDD</td>
<td>1, 35</td>
<td>.22</td>
<td>.64</td>
</tr>
<tr>
<td>3</td>
<td>Control, MDD, Dysthymia</td>
<td>2, 48</td>
<td>.33</td>
<td>.72</td>
</tr>
</tbody>
</table>

b. Valence main effects

<table>
<thead>
<tr>
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<th>Groups</th>
<th>d.f.</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Control, MDD</td>
<td>2, 54</td>
<td>.10</td>
<td>.91</td>
</tr>
<tr>
<td>2</td>
<td>Control, MDD</td>
<td>2, 48</td>
<td>1.05</td>
<td>.36</td>
</tr>
<tr>
<td>2</td>
<td>Control, MDD, Schizophrenia</td>
<td>2, 60</td>
<td>4.74</td>
<td>.03</td>
</tr>
<tr>
<td>3</td>
<td>Control, MDD</td>
<td>2, 70</td>
<td>1.37</td>
<td>.26</td>
</tr>
<tr>
<td>3</td>
<td>Control, MDD, Dysthymia</td>
<td>2, 96</td>
<td>.81</td>
<td>.45</td>
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</tbody>
</table>

c. Group x valence interaction

<table>
<thead>
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<th>Groups</th>
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<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Control, MDD</td>
<td>2, 54</td>
<td>1.41</td>
<td>.25</td>
</tr>
<tr>
<td>2</td>
<td>Control, MDD</td>
<td>2, 48</td>
<td>2.50</td>
<td>.09</td>
</tr>
<tr>
<td>2</td>
<td>Control, MDD, Schizophrenia</td>
<td>4, 60</td>
<td>5.03</td>
<td>.01</td>
</tr>
<tr>
<td>3</td>
<td>Control, MDD</td>
<td>2, 70</td>
<td>.13</td>
<td>.87</td>
</tr>
<tr>
<td>3</td>
<td>Control, MDD, Dysthymia</td>
<td>4, 96</td>
<td>.28</td>
<td>.89</td>
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</tbody>
</table>

Stouffer Meta-Analytic Values and Standard Deviations in Parentheses

<table>
<thead>
<tr>
<th></th>
<th>Major Depression</th>
<th>Dysthymia</th>
<th>Schizophrenia</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative words</td>
<td>0.08 (1.34)</td>
<td>0.36 (1.53)</td>
<td>-1.72 (3.52)</td>
<td>0.04 (1.44)</td>
</tr>
<tr>
<td>Positive words</td>
<td>-0.01 (1.32)</td>
<td>0.23 (1.13)</td>
<td>0.92 (3.61)</td>
<td>-0.15 (1.54)</td>
</tr>
<tr>
<td>Neutral words</td>
<td>0.14 (1.40)</td>
<td>0.02 (1.35)</td>
<td>-1.04 (3.22)</td>
<td>0.09 (1.39)</td>
</tr>
</tbody>
</table>

Note: Value = 1.95 is statistically significant at 90% level.
Table 3

*N400 means (and standard deviations) by group and study*

<table>
<thead>
<tr>
<th>Study</th>
<th>Major Depression</th>
<th>Dysthymia</th>
<th>Schizophrenia</th>
<th>Controls</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Negative Words</td>
<td>2.58 (1.84)</td>
<td>n/a</td>
<td>2.69 (3.55)</td>
</tr>
<tr>
<td></td>
<td>Positive Words</td>
<td>3.12 (2.22)</td>
<td>n/a</td>
<td>1.91 (4.53)</td>
</tr>
<tr>
<td></td>
<td>Neutral Words</td>
<td>2.63 (2.92)</td>
<td>n/a</td>
<td>2.30 (2.48)</td>
</tr>
<tr>
<td>Study 2</td>
<td>Negative Words</td>
<td>2.34 (1.71)</td>
<td>n/a</td>
<td>-6.80 (1.68)</td>
</tr>
<tr>
<td></td>
<td>Positive Words</td>
<td>1.28 (2.05)</td>
<td>n/a</td>
<td>5.34 (1.03)</td>
</tr>
<tr>
<td></td>
<td>Neutral Words</td>
<td>2.41 (2.71)</td>
<td>n/a</td>
<td>-2.30 (1.10)</td>
</tr>
<tr>
<td>Study 3</td>
<td>Negative Words</td>
<td>2.07 (3.23)</td>
<td>2.55 (2.71)</td>
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<tr>
<td></td>
<td>Positive Words</td>
<td>1.98 (2.70)</td>
<td>2.28 (1.84)</td>
<td>n/a</td>
</tr>
<tr>
<td></td>
<td>Neutral Words</td>
<td>2.42 (2.42)</td>
<td>2.38 (2.36)</td>
<td>n/a</td>
</tr>
</tbody>
</table>
Figure 1. Grand-average waveforms from Pz for Study 1, 2, and 3. Y axis is in microvolts. X axis is in milliseconds. Waveforms include a 200 pre-stimulus baseline subtraction. Zero is onset of the terminal, emotional word.