Brief psychological intervention for depression – An ERP study

Hanna Vanhatalo

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University of Jyväskylä
Department of Psychology
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ABSTRACT

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Author: Hanna Vanhatalo
Supervisor: Piia Astikainen
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Visual mismatch negativity (vMMN) is an event-related potential (ERP) component, which occurs when there is a change in visual environment. Previous studies have suggested that vMMN is elicited to several stimulus types, also to facial expressions. N170 is a widely studied ERP component which is typical in facial recognition. The purpose of this study was to find out if there was a vMMN in detecting neutral, happy and fearful expressions among non-depressed and depressed subjects. In addition, the effects of short psychological intervention to depressed subjects were examined. The oddball paradigm was used. The subjects watched pictures of neutral, happy and fearful facial expressions with changing identities. The probability for neutral expressions, i.e. standard stimulus, was 0.8 and for happy and fearful expressions, i.e. deviant stimuli, was 0.1 for both. The visual mismatch negativity was assumed to be elicited to changes in expressions. The depressed group also got a psychological intervention which included four therapy sessions with the means of acceptance and commitment therapy. The healthy, non-depressed control group consisted of 17 subjects. The depressed subjects were divided into two different groups, experimental and waiting list control groups. Both experimental and waiting list control group consisted of 23 subjects. The experimental group got the intervention immediately and waiting list control group after the first group. The event-related potentials were recorded using 14 electrodes placed to their skull, according to international 10-20 system. The EEG measurement was done once to non-depressed group, twice to experimental group and three times to waiting list control group. The vMMN elicited differently in non-depressed and in depressed group. It was found out that among non-depressed group, the vMMN elicits in consequence of happy expressions at P8 and fearful expressions at P7 and P8. In depressed group, there were no such effects. Happy expressions elicited larger responses at P8 than at P7 in the non-depressed group. In depressed group the responses were eliciting bilaterally, as well as the fearful expressions in both groups. The treatment effects were examined and it appeared that the responses to happy expressions diminished in experimental group after intervention. The same effect was not found in waiting list control group which had not received the intervention yet. When the experimental and waiting list control group were combined, the significant effects were found at P7 in fearful expressions. The results support the previous findings which have suggested that clinical states affect visual preattentive processing.

Keywords: facial recognition, visual mismatch negativity (vMMN), electroencephalography (EEG), event-related potentials (ERPs), depression, N170

TIIVISTELMÄ

Lyhyt psykologinen interventio masennuksen hoitoon - Herätevastetutkimus

Tekijä: Hanna Vanhatalo
Ohjaaja: Piia Astikainen
Psykologian pro gradu-tutkielma
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Visuaalinen poikkeavuusnegatiivisuus (vMMN) on herätevastekomponentti, joka ilmenee, kun visuaalisessa ympäristössä tapahtuu muutoksia. Aikaisemmat tutkimukset ovat osoittaneet, että vMMN syntyy useisiin ärsyketyyppeihin, myös kasvojenilmeisiin. N170 on laajasti tutkittu herätevastekomponentti, joka on tyypillinen kasvojen tunnistamisessa. Tämän tutkimuksen tarkoitus oli selvittää, syntyykö visuaalista poikkeavuusnegatiivisuutta ei-masentuneiden ja masentuneiden koehenkilöiden keskuudessa katsellessa neutraaleja, iloisia ja pelokkaita kasvonilmeitä. Lisäksi tutkittiin lyhyen psykologisen intervention vaikutuksia masentuneisiin koehenkilöihin. Tutkimuksessa käytettiin oddball-paradigmaa. Koehenkilöt katselivat valokuvia, jotka esittivät neutraaleja, iloisia ja pelokkaita kasvonilmeitä. Valokuvissa esiintyvien henkilöiden identiteetit vaihtelivat. Todennäköisyys neutraaleille ilmeille eli standardille ärsykkeelle oli 0.8 ja iloisille sekä pelokkaille ilmeille, poikkeaville ärsykkeille, 0.1 molemmille. Visuaalisen poikkeavuusnegatiivisuuden oletettiin syntyvän vaihteleviin ilmeisiin. Masentuneiden ryhmä osallistui lisäksi psykologiseen interventioon, joka koostui neljästä terapiakerrasta, jossa käytettiin hyväksymis- ja omistautumisterapian menetelmiä. Terveiden, ei-masentuneiden ryhmässä oli 17 koehenkilöä. Masentuneet koehenkilöt jaettiin kahteen eri ryhmään, koe- ja jonotuslistaryhmään. Sekä koe- että jonotuslistaryhmässä oli molemmissa 23 koehenkilöä. Koeryhmä sai intervention välittömästi, jonotuslistaryhmä koeryhmän terapian päätyttyä. Aivojen herätevasteita mitattiin neljällätoista elektrodilla, jotka asetettiin koehenkilöiden kallon pinnalle noudattaen kansainvälistä 10-20-järjestelmää. EEG-mittaus tehtiin kerran ei-masentuneiden ryhmälle, kahdesti koeryhmälle ja kolmesti jonotuslistaryhmälle. vMMN ilmeni erilaisena masentuneilla ja ei-masentuneilla. Havaittiin, että vMMN syntyi iloisiin kasvonilmeisiin elektrodilla P8 ja pelokkaisiin ilmeisiin sekä elektrodilla P7 että P8 ei-masentuneiden ryhmässä. Masentuneiden ryhmässä vastaavaa efektiä ei ollut. Iloiset ilmeet synnyttävät suuremmat vasteet P8:lla kuin P7:lla ei-masentuneiden ryhmässä. Masentuneiden ryhmässä vasteet esiintyivät bilateraalisesti, kuten myös vasteet molemmissa ryhmissä pelokkaiden ilmeiden tapauksessa. Tutkittaessa hoidon vaikutuksia ilmeni, että vasteet iloisiin ilmeisiin pienenivät koeryhmässä intervention jälkeen. Samaa vaikutusta ei löytynyt jonotuslistaryhmästä, joka ei ollut vielä saanut interventiota. Kun koe- ja jonotuslistaryhmät yhdistettiin, merkitseviä eroja havaittiin elektrodilla P7 pelokkaissa ilmeissä. Tulokset tukevat aiempia havaintoja, joiden mukaan erilaiset kliiniset tilat vaikuttavat visuaaliseen esitietoiseen prosessointiin.

Avainsanat: kasvojen tunnistaminen, visuaalinen poikkeavuusnegatiivisuus (vMMN), elektroenkefalografia (EEG), herätevasteet (ERPs), masennus, N170

CONTENTS

1. INTRODUCTION

Major depression is the most common mood disorder in all age classes. Its prevalence in Finland is about 5 % (Lönnqvist et al., 2007). According to the study of Isometsä, Aro & Aro (1997), the prevalence of major depressive episode is 4,3 %. Depression can be described as a diverse feeling, symptom, syndrome or illness which causes considerate suffering. Behavioural passivity, negative beliefs about the self, sadness, loss of interest, pessimism, changes in sleep, appetite and sexual interest and suicidal thoughts can be considered as the symptoms of depression (DeRubeis, Siegle, & Hollon, 2008). According to DSM-IV, depression can be subdivided into major depressive disorder and dysthymic disorder (Heller & Nitschke, 1998). Depression is highly common in worldwide so it is significant to try to understand and prevent it. In Finland, major depressive disorder is more common among females than males (Isometsä et al., 1997) and it has a high comorbidity with other psychological disorders (DeRubeis et al, 2008). There are many causes for depression. Depressive episodes may be a result of medical or neurobiological illnesses, psychiatric disorders or pharmacological agents. Also stress, work-demands and interpersonal interactions are playing a big role in the development of depression. (Drevets, 2001). Major depression has a significant effect on social relationships and occupational functioning so it is a remarkable reason for disablement (DeRubeis et al., 2008). It is noticeable, that only about 50 % of those suffering depression perceive the need for treatment (Isometsä et al., 1997). The vast majority will not contact mental health services which has an impact on the whole society.

Mineka and Sutton (1992) suggest that there is a memory bias for negative mood-congruent material in depression. Depression is associated with ”looking back” rather than planning the future. Negative self-evaluations and loss are filling the thoughts of depressed person. Associative biases may play a strong role in the progression of depression (Mineka & Sutton, 1992). Negative self-evaluations, expectancies and memories are the underlying themes in depression (Beck, 2008). Beck (2008) is suggesting there is a comprehensive cognitive model of depression, which consists of two different levels: negative self-reports and systematic cognitive bias. Negative schemas result in the apparent symptoms of depression like sadness, loss of

motivation, regressive behaviour and inactivity. These factors lead to negative reinforcing loop. Beck (2008) is proposing that genetic vulnerability may lead to the development of depression.

It is obvious that the depression is not only the disorder of behaviour but also the brain. Emotion and brain activity are closely linked together and cannot be evaluated alone. Depression has an impact on basic instincts that affects everyday functioning, like self-preservation, maternity, sexuality and experiencing pleasure. Furthermore, biological functions are also disturbed. These consequences give a good reason to explore which phenomena are affecting in the major depression. (Beck, 2008).

DeRubeis et al. (2008) state in their article that many studies has proved the effectiveness of cognitive psychotherapy in treating major depression. Therapy has the same effects as the medication, in addition cognitive therapy reduces more the risk of relapse. If people stop taking their medicines too early can cause a relapse in some cases. The use of mere antidepressants is a risk, too. Dobson (1989) points out the superiority of cognitive therapy compared with a waiting list, no-treatment control, medicine and different psychotherapies. Clients treated with cognitive therapy did better than 70 % of drug therapy patients (Dobson, 1989). Previous studies have revealed the effectiveness of psychotherapy in treatment of depression – it is possible to verify the effects of psychotherapeutical intervention by using neuroimaging (Roffman et al., 2005). It is suggested that different treatments affect the same neural mechanisms, probably limbic and prefrontal circuitry, although with different pathways. DeRubeis et al. (2008) propose that cognitive therapy affects especially on the prefrontal function while antidepressants have on effect on amygdala. However, it seems that cognitive therapy produces effects that antidepressant does not because the treatment effect is enduring. Many variables such as genetic, patient history, cognitive and symptom has an impact on the effectiveness of psychotherapeutical or medical treatment. There are reversible studies about the effects of psychotherapy in comparison to medical treatment according to the review article of Roffman et al. (2005). In one study, metabolism in frontal regions diminished due to cognitive-behavioral therapy. With medication, metabolism increased so it works through different mechanisms comparing to therapy. In another study, cognitive-behavioral psychotherapy caused increases in activity in the ventral frontal and subcortical areas.

Interpersonal psychotherapy has the same effects. Using medical treatment, the activity decreases in dorsal frontal regions. It is suggested that psychotherapy works by using top-down processing mechanisms while pharmacotherapy can be considered as bottom-up treatment (Roffman et al., 2005). Despite these observations, there is still much to explicate in these mechanisms.

Generally, the effectiveness of psychotherapy has been studied based on the survey data and subjective assessment. It would be essential to study the role of the brain activity in different clinical states, in this case the major depression. It is an indisputable fact that the various brain regions are determining the human behaviour and mood. Each region of the brain has a responsibility in processing particular information. (Heller & Nitschke, 1998). There is evidence that assessment of brain function can be used as a predictor for depression onset and recurrence (DeRubeis et al., 2008).

1.1. Brain activity and face perception in depressed patients

Mood and neurobiological aspects are closely linked together although the connection is not very well defined. There is no specific location for emotions in the brain but several brain areas are corresponding for regulation of emotions (Heller & Nitschke, 1998). Heller and Nitschke (1998) among many other researchers assume that diminished activation of the right hemisphere is related to depressed mood states. Although the role of the right hemisphere in depression is proved in many studies, there are no findings of higher left hemisphere activation in unipolar depression (Heller & Nitschke, 1998). Many studies have suggested that the decreased right-posterior activity is related to depression. (Bruder et al., 1995; Heller & Nitsche, 1998; Deldin et al., 2000). The same kind of phenomenon is seen in the case of brain damage. The damage in the right hemisphere causes euphoric reaction compared to left hemisphere damage which externalizes in depressed mood and crying (Heller & Nitschke, 1998). The underlying explanation is still vague. However, the impact of hemispheres is quite inconsistency according to certain studies. Nevertheless, there is convincing evidence that diminished right parieto-temporal activity is associated with suppressed right hemisphere function and depression (Heller & Nitschke, 1998).

There is evidence from brain-imaging and post-mortem studies that depressed patients have abnormalities in their brain function and structures comparing to healthy persons (Leppänen et al, 2004). It is suggested that frontal EEG asymmetry is associated with vulnerability to depression. The asymmetry may also be a sign from previous depressive episode (Gotlib, Ranganatha, & Rosenfeld, 1998). Functional neuroimaging has revealed that amygdala plays a great role in perception of emotions, also in facial recognition (Zhao & Li, 2006). The elevated activity of amygdala is associated with depressed mood. Also prefrontal cortex participates in altering emotional behaviour. (Drevets, 2001; Cook et al., 2002, Beck, 2008). Hypersensitive amygdala is a risk for depression because it is related to genetic factors and negative cognitive bias and may work as a neurophysiological correlate of cognitive bias (Beck, 2008). Also, these aspects combined with diminished function of prefrontal lobe is associated with depression (Beck, 2008). Cook et al. (2002) suggest that brain activity in prefrontal regions alters due to antidepressant medications. Early changes in that area are associated with the completeness of clinical response according to Cook et al (2002). Brody et al. (2001) investigated changes in symptoms and in regional brain metabolism after treatment with either medicine (paroxetine) or interpersonal psychotherapy. They found out that anxiety and sadness has a connection with brain metabolic change along the treatment. Especially limbic-cortical regions are affecting to major depressive disorder. There is no doubt that treatment could not affect the function of the brain.

The judgement of emotions play a remarkable role in depression (Hale, 1998). There is evidence that patients with depression may have a bias in their information processing and cognitive functions (Bouhuys, Geerts, Mersch, & Jenner, 1996; Deldin et al., 2000; Astikainen, 2008). Especially emotional stimuli are judged differently by depressed than people with no depression. Hale (1998) reports that depressed patients judged facial expressions more negatively than non-depressed persons did. This may lead to difficulties in interpersonal relationships and may be a significant factor in depression persistence. It is hypothesized that the processing of emotional stimuli may be a permanent trait which would explain the high probability to depression recurrence (Leppänen et al, 2004). The study of Leppänen et al. (2004) suggested that depression affects more on the recognition of neutral faces than the recognition of happy and sad faces. Bouhuys et al. (1996) state that depression is more persistent among people who

are hyposensitive to negative facial stimuli. Unpleasant social experiences may lead to negative consequences and enhance the progression of depression. Neural functioning has a great role in reactivity to negative experiences (Beck, 2008). Bouhuys et al. (1996) are also pointing out that the inaccurate perception of negative facial expressions may be due to deficits in right hemisphere functioning. DeRubeis et al. (2008) are suggesting that the continuous negative thinking is associated with increased activity in limbic regions and decreased activity in the prefrontal cortex. The activation of the prefrontal cortex is examined with resting-state electroencephalograms which indicates the reduced activity in left prefrontal cortex among depressed individuals (Davidson, 2003). Shestyuk et al. (2005) investigated the brain activity during processing of emotional stimuli with depressive subjects and found out that there was a decrease in activity during processing of positive information. Strengthened processing of negative stimuli can cause biases in behavioral and physiological responses such as lack of reactivity to positive events. In the study of Kayser et al. (2000), depressed subjects did not show elevated brain activity to negative compared to neutral stimuli whereas the negative stimuli were eliciting larger event-related potentials compared to neutral stimuli among non-depressed control subjects. They suggest that the selective inhibition of right parietal regions is the cause for diminished processing in the context of evaluating emotional stimuli.

1.2. The face-specific N170 component

Event-related potentials reflect electrical activity of the brain as a consequence of experimental manipulation and certain stimuli. Sensory processes generate various ERP-components. Repetition is needed in revealing event-related potentials. Event-related potentials are divided into endogenous and exogenous components. Endogenous components originate as a result of internal stimulus. Exogenous components elicit by the external stimulus and they are quite stable. (Näätänen, 1992).

Face perception is investigated widely in the field of cognitive neuroscience. Especially electrophysiological experiments have proven some fundamental information about humans' ability to perceive faces of other human beings.

Electroencephalography (EEG) and especially event-related potentials (ERPs) are used in this kind of studies. Evidence of a face specific event-related potential was found in a study of Bentin et al. (1996). They suggest that human faces evoke a negative potential at 172 ms. The component is called N170 and it is especially sensitive for human eyes. It was not elicited by other objects like animals, human hands, cars or scrambled faces. The amplitude of N170 was larger on the right than in the left hemisphere in healthy subjects. (Bentin et al., 1996) The existence of remarkable factor in human information processing and face perception is proved by this and several other studies. In the study of Batty and Taylor (2003), ERPs were recorded while presenting six basic emotions and neutral expressions. They found out that N170 was seen around 140 ms and N170 to fear, disgust and sadness endured longer than N170 to happy and neutral faces. Besides, fear evoked larger N170 than other emotions. Those ERPs also elicited later. In another study, N170 amplitudes were larger for upright and inverted faces than other object categories (Itier & Taylor, 2004). They also suggest that N170 amplitude was significantly larger over the right than the left hemisphere. Posterior parietal electrodes P7 and P8 were used. Deldin et al. (2000) are presenting previous research which states that right hemisphere processes the vast majority of face perception. In addition to component N170, also N200 is used in examining face recognition because of its specificity to face perception (Deldin et al., 2000). They found out that N200 in the right-posterior region was reduced in the group of depressed subjects. Thus, these abnormalities in brain activity may have an impact on mood and cognitive processing in the major depression.

1.3. Visual Mismatch Negativity (vMMN)

Mismatch negativity in the auditory modality is negative event-related component (ERP) which elicits when there is a change in the acoustic environment. It generates 150-250 msec after the occurrence of the deviant stimulus (Näätänen, 1992; Pazo-Alvarez, Cadaveira, & Amenedo, 2003). MMN is often studied with the oddball paradigm wherein a high-probability standard stimulus is occurring with infrequent deviant stimuli. The oddball paradigm can be passive or active, however MMN is best observed especially in non-attentional condition (Pazo-Alvarez et al., 2003). MMN elicits to the infrequent stimulus. Consequently, it cannot be elicited by itself; at least

two different stimuli are always needed. The attention is not needed in the elicitation of MMN therefore it has biological importance as an alert sign because there is something in the environment which does not match expectations (Näätänen, 1992). MMN indicates the function of memory traces in the brain and it generates frontocentrally, being larger in the right hemisphere (Pazo-Alvarez et al., 2003).

The existence of mismatch negativity in visual modality is more controversial than auditory mismatch negativity. Nevertheless, the recent results have shown that vMMN exists and can be compared to its auditory counterpart in some features. Visual mismatch negativity is associated with automatic and preattentive visual information processing and it works as a precursor of attention (Urban et al., 2008, Pazo-Alvarez et al, 2003). Pazo-Alvarez et al. (2003) present in their review several studies suggesting that vMMN elicits as a response in various stimuli characteristics such as direction of movement, colour, size, location, contrast, orientation, spatial frequency and form. What is essential is that the procession has to be occurred automatically, without attention (Tales, Newton, Troscianko, & Butler, 1999). vMMN is studied with oddball condition although bigger difference between standard and deviant stimuli are needed than in auditory MMN (Pazo-Alvarez, 2003). Visual MMN distributes over occipital and posterior temporal cortex, hence the visual areas are significant for the elicitation of vMMN (Tales et al., 1999). Tales et al. (1999) made a conclusion of the characteristics of visual MMN: attention is not needed, it is evoked automatically and it originates from visual association cortex. Visual MMN reflects the pre-attentional and automatic information processing therefore auditory and visual MMN have the same function. There is evidence from several studies that the latency of vMMN is varying between 150 and 350 ms after stimulus. Visual mismatch negativity is studied with both intermodal and intramodal experiments. There is also right hemisphere dominance in the elicitation of vMMN. (Pazo-Alvarez et al., 2003).

Visual modality is not very sensitive to changes that occur in non-attentional condition. That phenomenon is called change blindness. However, some authors point out that visual system can register the change without consciousness. Visual and auditory systems may have similar underlying mechanisms considering the pop-out phenomena, which is a stimulus that catches the attention over distractors. Unless there is no pop-out, it would not be possible to detect unexpected information. (Pazo-

Alvarez et al., 2003). Pazo-Alvarez et al. (2003) are suggesting that it would be important to find out how the dorsal and ventral streams in visual processing are affecting to elicitation of vMMN.

A study by Fu, Fan and Chen (2003) revealed a change-related P192 as a candidate to visual MMN. They are proposing that a visual sensory memory trace can be elicited more easily than its auditory counterpart can. That is because a visual memory can be formed without a repetition and changes in the context (Fu et al., 2003). As other studies, Fu el al. (2003) found also a right-hemisphere dominance of supposed vMMN.

It is suggested that rareness rather than mismatch is associated with visual change detection. The assumption was based on the occurrence of vMMN: if there is a mismatch in response to deviants among standards and also to rare standards, the phenomena can be caused more as an implication of rareness rather than mismatch. (Tales et al, 1999; Kenemans, Jong, & Verbaten, 2003). Kenemans et al. (2003) are using term rareness-related negativity (RNN) instead of mismatch negativity and suggesting that the rarity has a neurophysiological implementation; neurons habituate to the standard stimulus and when there is a deviant stimulus with longer intervals, the neurons are corresponding more intensely because the habituation has not occurred. Mismatch response requires additionally a memory trace of the past stimuli.

Zhao & Li (2006) were first to detect a MMN in unattended condition using facial expressions as a stimulus unlike many other studies which have used for example geographic patterns as a stimuli. That might be a sign from the automatic processing of face-related material. The healthy subjects were reacting to tones pressing the button. Because their attention was centered to auditory modality, they ignored the facial expressions which were seen on the screen. The faces were selected from *Pictures of Facial Affect* (Ekman & Friesen, 1976). The face-specific component called N170 was seen especially in occipito-temporal sites. Zhao & Li called their finding as expressional mismatch negativity (EMMN) and it was larger to sad faces than happy faces. They assumed that larger deviances are needed to elicit visual mismatch negativity than auditory mismatch negativity. (Zhao & Li, 2006).

Visual mismatch negativity in the context of facial recognition is quite recent area of research and the used paradigms have limitations. E.g. Zhao & Li (2006) used pictures of one person only in their study. Astikainen & Hietanen (2009) studied vMMN in facial expressions using pictures with varying facial identity. An oddball paradigm was used. The subjects were engaged to an auditory task while seeing fearful (deviant), happy (deviant) and neutral (standard) expressions on the screen. The occipital negativity was found at the latency of 150-180 ms, which is in line with previous findings (e.g. Pazo-Alvarez et al., 2003). In addition, happy deviants were detected earlier than fearful deviants were. Fearful deviants also elicited smaller responses than happy deviants did.

Recent study has shown that there are significant differences in the vMMN in different clinical groups: among patients with schizophrenia, the amplitude of vMMN was significantly smaller than in healthy control subjects. The conclusion may be thought as an impairment of automatic preattentive processing of sensory information in schizophrenia. Visual MMN may be a precursor of a progression in schizophrenia. The connection of cognition and vMMN has not been proved yet. However, auditory MMN and cognitive processes are closely linked to each other. (Urban et al., 2008). Tales et al. (2008) have studied the connection of mild cognitive impairment, Alzheimer’s disease and vMMN. Alzheimer's disease and mild cognitive impairment (MCI) has a significant effect on cognition, memory, perception, language and executive functions (Tales et al., 2008). It would be useful to uncover the factors that precede these diseases. Alzheimer's disease and in MCI has a debilitating impact on visual selective attention hence vMMN could be a suitable indicator to these impairments. Tales et al. (2008) state that the change-detective vMMN diminishes during the course of healthy ageing while in Alzheimer's disease and in MCI the decrease is remarkable.

Based on previous studies of different clinical groups, could visual mismatch negativity determine difficulties in preattentive emotional processing in the case of major depression? That is what we were trying to find out. There is evidence that the component N170 could be suitable counterpart of vMMN, therefore it is used in this study as well.

1.4. Acceptance and commitment therapy

Acceptance and commitment therapy (ACT) is one of the new ”third-generation” therapies which basis is on philosophy called functional contextualism and also Relational Frame Theory (Hayes & Batten, 1999). Its founder is Steven C. Hayes. ACT is based on an assumption that life is full of suffering which we are trying to control, usually with the help of language. However, language is not the best way to control thoughts and feelings because those are impossible to control. Consequently, the aim is to accept those thoughts and feelings instead of suppressing them. (Lappalainen et al., 2004). ACT uses various experiential exercises such as metaphors, working with values and exercises which are increasing consciousness skills. The goal is to produce psychological flexibility (Lappalainen et al., 2007).

Previous studies have shown that acceptance and commitment therapy is an effective treatment for different psychological problems. In the study of Dahl, Wilson and Nilsson (2004), acceptance and commitment therapy is used quite successfully in the reduction of stress and pain symptoms. Therapy treatment has better outcomes than medical treatment. Also McCracken, Vowles and Eccleston (2005) found out that acceptance-based therapy improved emotional, social and physical functioning among patients with long standing chronic pain. Acceptance and commitment therapy is also used in the treatment of drug addiction (Hayes et al., 2004). Therefore, acceptance-based therapies are used in many kind of situations. In this study, we are trying to find out the effectiveness of ACT in the concept of major depressive disorder.

1.5. Hypotheses

The purpose of this study is to describe the phenomena of N170 and visual mismatch negativity in the context of face perception among depressed and non-depressed subjects. We are also interested in the effectiveness of short therapeutical intervention using methods of ACT and its affects to the brain activity, more closely to the visual mismatch negativity which is a component of event-related potentials. There is evidence that the intervention has an impact in patients’ mood. The self-evaluation about the mood was considerably more positive after the therapy as well as the score

of Beck's Depression Inventory and Symptom Checklist. Is it possible to detect the impairment in mood also in the brain function?

In this study, we wanted to find out whether the information processing in depressed subjects differs in coding the structure of the face and other visual traits (N170) or in change perception considering the facial expression (vMMN). Visual mismatch negativity is a difference between the responses to standard stimuli and responses to infrequent deviant stimuli (Näätänen, 1992). Happy and fearful expressions are expected to elicit bigger amplitudes than neutral standards. However, amplitude differences between happy and fearful are not expected (see also Astikainen & Hietanen, 2009). This study is a part of the research of the Department of Psychology, University of Jyväskylä.

2. METHOD

2.1. Participants

There were 46 depressed subjects in this study. The depressed subjects were randomized into two groups, the other got the intervention immediately and the other were on a waitlist and got the therapy after the experimental groups' measurements. The sample of 17 non-depressed control subjects was recorded and used as a comparison in this study. All of the subjects were right-handed and had normal or corrected-to-normal vision. Depressed patients were recruited with the aid of an advertisement in a local newspaper. They made a contact by phone or by e-mail and told they wanted to take part of the project. The therapy was free for them. The non-depressed control group consisted for example the students from Open University and other communities. What was common to the non-depressed control subjects is that they did not consider themselves as depressed and there were no evidence of mood descendance in survey data. EEG-recordings were done once to the non-depressed controls, twice to the experimental group and three times to the waiting list control group. The survey data was collected before and after therapy period from the experimental group and from the waiting list control group. The waiting list control group filled in the surveys also after the waiting period. Non-depressed control subjects filled in the surveys only once. The mean age of subjects in the depressed group was 46,4 years ranging between 28 and 61 years. The standard deviation of the subject's age was 9,867. In the healthy control group, the mean age was 45,5 years ranging between 30 and 58 years. The standard deviation was 7,908. There were 38 women and 8 men in the experimental group and 14 women and 3 men in the control group. All subjects gave written informed consent.

2.2. Therapy and therapists

In this study, we used the methods of acceptance and commitment therapy as an intervention to depressed patients. The content of every therapy session was planned beforehand so the main structure was the same among all therapists and patients. The exercises, for example mental practices, varied according to patient's situation. Four

sessions of therapy (á 60 minutes) were provided to experimental and waiting list control groups by psychology students and weekly supervision was received. The psychology trainee therapists were doing their masters' thesis and/or vocational studies during the therapy period.

2.3. Survey data

The results of two different inventories (Beck's Depression Inventory and Symptom Check List-90) were especially the targets of interest in this study. Inclusion criterion to the depressed groups was that the client had more than 10 points in the BDI. The mean score of BDI in experimental group was 26.35 and in waiting list control-group 23.22. The mean of healthy controls' scores was 1.63. Mean scores in SCL-90 were 1.49, 1.10 and 0.08 respectively. In addition, the subjects filled in other inventories like AAQ-2, KIMS, JES, BAI and Ojanen's Mood, Self-confidence and Satisfaction to life which measure mindfulness and anxiousness, for example. All of the participants filled in the surveys. There were also a diagnostic interview to the subjects based on DSM-IV-TR criteria.

2.4. EEG recording

Electroencephalography was recorded with Brain Vision Recorder software. The data was collected with Ag-AgCl electrodes from the F3, Fz, F4, C3, Cz, C4, P3, Pz, P4, P7, P8, O1, Oz and O2 according to the international 10-20 system. Two electrodes, one above the left eyebrow and another on the right outer canthus recorded the eye movements. Although average reference was used in the final analysis, electrodes placed in both mastoids served as a reference as well. Impedances were maintained below 5 kΩ. The sampling rate of the data was 1000 Hz. The online low-pass filter was set to 100 Hz allowing frequencies under 100 Hz to pass. Offline filter was set to 30 Hz. Electro-oculography was recorded to detect ocular artefacts which were removed using Gratton-Coles method as an ocular correction. Data was segmented to -800 - 700ms, and 100 ms before stimulus onset was used as a baseline. If larger than ±50 µv artefact were detected on any electrode, the data was deleted 200 ms before and 200 ms after artefact.

2.5. Stimuli and paradigm

The paradigm was a modification of the oddball paradigm in which two deviant types were applied. In the paradigm, a standard stimulus was repeated in high probability with a deviant stimuli appearing with smaller probability. The oddball paradigm were passive hence the subjects were listening to a radio play while steering his/her gaze to the 15´ computer screen placed one meter in the front of the subject. Visual stimuli were black and white pictures of human faces with neutral, happy and fearful expressions of four different models (female actors NR and PF, male actors EM and JJ from *Pictures of Facial Affect* by Ekman & Friesen, 1976). The standard stimuli were neutral expression, happy and fearful were deviant stimuli. Neutral facial expressions were presented 1600 times while both happy and fearful expressions were presented 160 times. The probability for the standard stimuli was 0.8 and for both deviants 0.1. The interstimulus interval (ISI) of the stimuli was 500 ms. The facial expressions were seen on the screen during 200 ms. The duration of the whole oddball task was 20 minutes on average. The subjects were ignorant of the specific questions investigated.

2.6. Data analysis

Vision Analyzer 2.0 was utilized in ERP analysis. First, the EEG data was filtered with the low-cut off of 0,1 Hz and high cut off of 30 Hz and after that there were an ocular correction (Gratton-Goles method). The data was segmented into smaller items and the peak values were measured. Segmentation was set to start at 800 ms before and end 700 ms after the stimulus onset. Notch filter cut off the artefacts caused by mains current. The analysis consisted the peak values from two channels, P7 and P8 in the time window 125-195 ms after the stimulus onset, corresponding to the expected latency of used component N170 (Bentin et al., 1996). The difference between neutral (standard) and happy or fearful (deviants) expressions represent the vMMN component. The above-mentioned channels have been chosen because they are situated in the back of the skull, in the area of parietal and occipital lobe which is considered to be remarkable in face perception and the elicitation of vMMN. Itier & Taylor (2004) found out that N170 amplitude was larger in the right hemisphere in posterior parietal electrode (P8). The effect was not seen in the left hemisphere, P7.

The vMMN is usually elicited 100-200 ms from stimulus onset (Pazo-Alvarez et al., 2003) and we are using the component N170 representing the vMMN. Finally, the data was imported to SPSS software (Statistical Package for Social Sciences, version 15.0 for Windows) which was used in analysis.

Data were compared using a repeated measures analysis of variance (ANOVA) and Student t-tests. First, the differences between depressed and non-depressed subjects in face perception and ERP responses were analyzed. Finally, the analysis considering the effects of the intervention was accomplished, concentrating on the ERP component N170. Repeated measures of analysis of variance was used because the stimuli were presented sequentially and the standard stimuli were compared to deviant stimuli. Also the changes over time were the target of interest. In the case of significant effects, the Huynh-Feldt correction was utilized.

2.6.1. Stimulus trials

The number of accepted trials in EEG-paradigm were measured. In the non-depressed control group, the mean varied between 103-104 depending on the expression type. In the depressed group the range of mean values was 93-95 at the first measurement, 89-90 at the second and 73-75 at the third measurement depending on the expression type. There were no significant differences between trials per time of measurement in non-depressed group and in experimental group. In waiting list control group the difference was significant between first and second measurement in *fearful* (89 vs. 75 accepted trials) and between first and third measurement in *happy* (92 vs. 73) and in *fearful* (93 vs. 76). However, because the number of accepted trials decreased along the measurements, it may not favor possible treatment related effect.

3. RESULTS

3.1. Comparison of the depressed groups

The differences of the experimental and the waiting list control group were analyzed. An interaction was found in stimulus type × expression × electrode site × group, F(8.660)=44, p=0.005. Post hoc analysis revealed that there was a significant difference between responses to standard and deviant stimuli in fearful expressions at P7, t(-2.819)=22, p=0.010, and in happy expressions at P8, t(4.265)=22, p=0.001 in the experimental group. Standard and deviant stimuli also differed at P7 in both happy, t(2.928)=22, p=0.008, and fearful expressions, t(-2.512)=22, p=0.020 and at P8 in fearful expressions, t(-2.868)=22, p=0.009 in waiting list control group. That is to say, that the depressed groups did not have the same baseline in the beginning; the vMMN did not appear in same locations in both groups. Only the elicitation of vMMN at P7 to fearful expressions was common between groups. Other stimuli appeared differently at electrodes P7 and P8 in experimental and waiting list control group. This can cause problems in analyzing the data, however, we decided to make the groupwise analysis despite the variation on the baseline.

The experimental group and the waiting list control group did not differ in the scores of BDI questionnaire, t(1.322)=44, p>0.05. However, the groups differed significantly in SCL-90 scores, t(2.735)=44, p=0.009. This aforementioned significance means that SCL-90 scores cannot be considered as a reliable baseline in the assessment of therapy effects on behavioral indicators. On the contrary, BDI seems like a suitable tool for that purpose.

3.2. The existence of visual mismatch negativity (vMMN) and N170

3.2.1. Non-depressed vs. depressed subjects

The non-depressed and depressed groups were compared with 4-way ANOVA. Groups differed almost significantly from each other in expression × electrode site, F(3.510)=61, p=0.066. Because there was a main effect on stimulus type, F(35.547)=61, p=0.001, the variable was included in post hoc analysis, which was

decided to carry out despite the fact that the interaction was only almost significant. In non-depressed group, fearful expressions were processed at electrodes P7, t(-2.858)=16, p=0.011, and at P8, t(-2.692)=16, p=0.016, while happy expressions were processed only at P8, t(5.453)=16, p=0.001. In depressed group, ERPs to standards and deviants differed in both expressions and in both electrodes, happy at P7: t(3.508)=45, p=0.001, fearful at P7: t(-3.808)=45, p=0.001, happy at P8: t(3.452)=45, p=0.001 and fearful at P8: t(-3.214)=45, p=0.002. Because the interaction was almost significant, the non-depressed and depressed groups were analyzed separately. The figures 1 and 2 present the N170 responses to happy expressions in non-depressed and depressed subjects. As can be seen, the healthy control subjects elicited larger responses at P8 than at P7. The depressed subjects' responses were in the same size both at P8 and at P7. The figures 3 and 4 present fearful expressions. It seems that in non-depressed group, fearful expressions elicited larger responses at P8 than at P7.

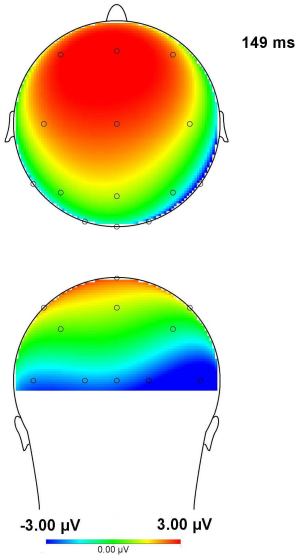

Figure 1. ***The non-depressed group.*** *N170 to happy expressions.*

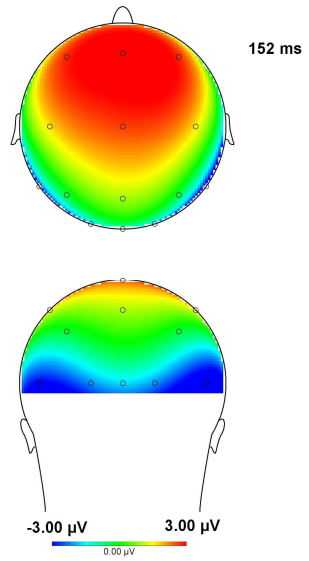

Figure 2. ***The depressed group.*** N170 to happy expressions.

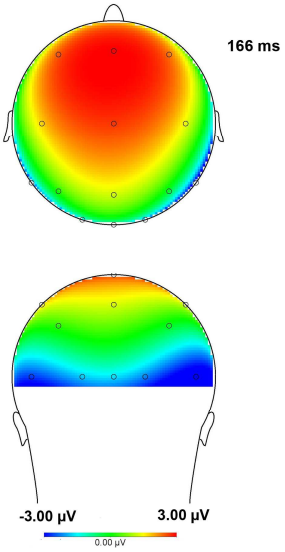

Figure 3. ***The non-depressed group.*** *N170 to fearful expressions.*

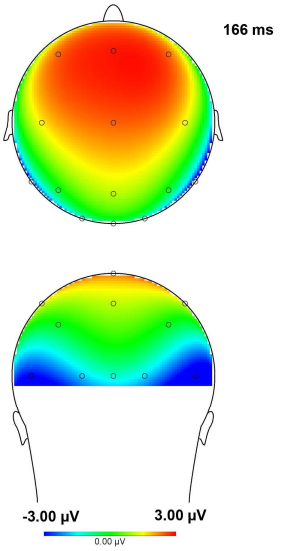

Figure 4. ***The depressed group.*** *N170 to fearful expressions.*

Paired samples t-tests were carried out to study further the differences between electrodes and vMMN. A significant difference between ERP responses to standard and deviant stimuli at electrodes P7 and P8 were found in depressed group; for vMMN at P7 $t(45)=4.125$, $p=0.001$ and at P8 $t(45)=4.145$, $p=0.001$. There was no large difference in the size of vMMN from electrodes (responses from deviants *happy* and *fearful* are combined): the mean amplitude at P7 was 0.56939 µV and at P8 0.49409 µV. The same analysis for non-depressed subjects revealed a significant difference between ERP responses to standard and deviant stimuli at electrodes P7 $t(16)=2.719$, $p=0.015$ and at P8 $t(16)=4.345$, $p=0.001$. Stimulus type discrimination, the mismatch negativity (standard vs. deviant), was higher for non-depressed subjects at P8, 0.73456 µV, than at P7, 0.43421 µV.

3.2.2. Non-depressed control subjects

First, the aim was to study the appearance of N170 in face perception and whether there was a visual mismatch negativity between standard and deviant stimuli. This hypothesis was tested in both non-depressed and depressed subjects. The correlations were checked and there were no significant effects between age and stimulus type, expressions or electrode sites.

In non-depressed, for face-sensitive ERP-component N170, a 3-way ANOVA, stimulus type (standard, deviant) × expression (happy, fearful) × electrode site (P7, P8), showed a significant main effects of stimulus type, $F(20.793)=16$, $p=0.001$ and electrode site, $F(24.019)=16$, $p=0.001$. Based on these main effects, the responses varied depending on the stimulus (standard or deviant) which means that the vMMN was elicited. In addition, the amplitudes of ERPs were significantly different depending on the electrode. Statistically significant interaction effects were found in expression × electrode site, $F(5.318)=16$. $p=0.035$, put differently, *fearful* and *happy* are processed differently in distinct locations. Stimulus type × expression × electrode site almost reached a level of significance, $F(3.316)=16$, $p=0.087$. This interaction can be considered as a trend.

Although the three-way-interaction was not statistically significant, post hoc

comparisons between stimulus type, expression and electrode site were carried out. Happy expressions did not elicit a vMMN at P7 area, t(1.227)=16, p>0.05, but at P8 there was an obvious vMMN, t(5.453)=16, p=0.001. Thus, the difference in ERPs between neutral and happy expressions were larger at P8 than at P7 in healthy control group. Figure 5 presents the vMMN responses to happy expressions. The visual mismatch negativity was significant only at P8 although there is no large difference on responses in the picture. However, happy expressions did not show a significant effect at P7. Fearful expressions elicited a vMMN at both P7, t(-2.858)=16, p=0.011, and P8, t(-2.692)=16, p=0.016.

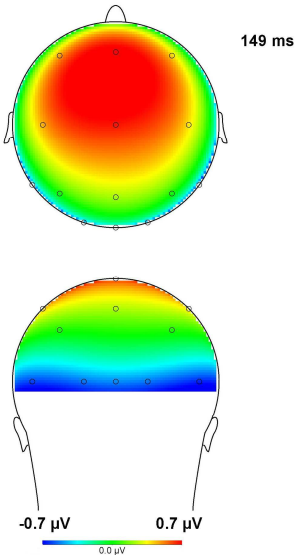

Figure 5. ***The non-depressed group.*** *vMMN to happy expressions. The visual mismatch negativity was significant only at P8.*

3.2.3. Depressed subjects

Depressed subjects had only the same main effects as the non-depressed subjects, stimulus type, F(26.523)=45, p=0.001 and electrode site, F(10.352)=45, p=0.002. Significant effects between the expressions were not found unlike in the non-depressed group. The Figure 6 shows that responses at P7 seems to be larger than at P8 but the difference was not statistically significant.

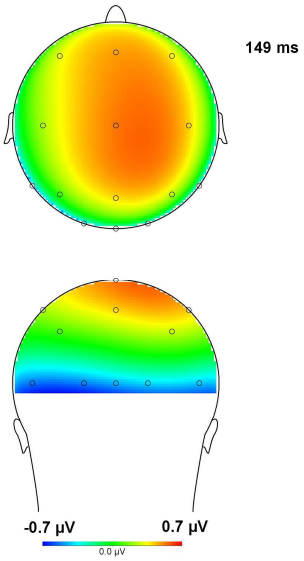

Figure 6. ***The depressed group.*** *vMMN to happy expressions. Although the responses seem to be larger at P7 than at P8, the effect was not significant in depressed subjects.*

3.3. ERP responses related to intervention

3.3.1. Experimental and waiting list control-groups

The analysis of the effects of the therapeutical intervention was started with comparing the experimental and the waiting list control-group in the time *pre* and *mid*, i.e. the time of measurement. *Pre* refers to the measurement before therapeutical intervention and *mid* to the second measurement when the experimental group have received the intervention and the waiting list control-group have not. In other words, the effects were studied in both groups but only the experimental group had been to therapy sessions. The groups differed in responses to expressions and time of measurement, $F(6.772)=44$, $p=0.013$. Almost significant trend effect was found in interaction between stimulus type × expression × electrode site × time of measurement × group, $F(3.568)=44$, $p=0.066$. The groups differed as regards all variables in the situation when one group had got therapy and other had not. There were significant differences between ERPs in experimental group. ERPs to happy expressions at P7 differed at the post-treatment measurement compared to pre-treatment measurement, $t(-2.472)=22$, $p=0.022$, as well as happy expressions at P8, $t(-4.126)=22$, $p=0.001$, and fearful expressions at P8, $t(-2.187)=22$, $p=0.040$. There were no significant differences in waiting list control group between ERPs and time of measurement.

Although the experimental and waiting list control group differed from each in the beginning, that is to say their baseline was not at the same level, more closely examination was made to reveal if there were some underlying effects.

3.3.2. The effects of intervention to experimental group

ANOVA was carried out for the experimental group which got the intervention between the time points *pre* and *mid.* The time of measurement was a variable in interaction with expression type (*happy, fearful*), F(9.633)=22, p=0.005. Since there were no effects for electrode site and stimulus type in ANOVA in the case of time of measurement, the variables were integrated for the post hoc comparisons. The responses from electrodes P7 and P8 were combined as one variable. In addition, the effect of stimulus type (standard vs. deviant) was eliminated and only the expression type (*happy, fearful*) was included in the post hoc analysis. In addition, a significant main effect was found in time of measurement, F(8.086)=22, p=0.009 and in stimulus type (vMMN), F(16.580)=22, p=0.001. Stimulus type, expression and electrode site showed a significant interaction, F(5.052)=22, p=0.035 therefore different stimuli elicited different ERPs at P7 and P8. However, this interaction did not include the time of measurement, which indicates about the treatment effect.

Post hoc comparisons between expression types were significant for happy, t(-3.756)=22, p=0.001. On the contrary, *fearful* had no effect, t(-1.261)=22, p=0.221. *Happy* also elicited larger visual mismatch negativity responses (mean for *happy* -0.62101; *fearful* -0.19142). Figure 7 show the N170 responses to happy expressions. The responses differed significantly after the intervention.

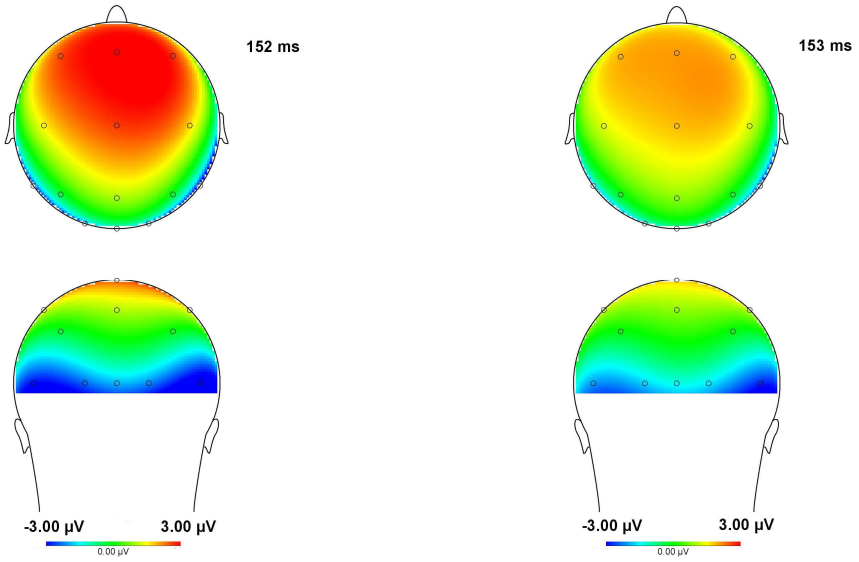

Figure 7. ***Pre- and post-treatment measurements in the experimental group.*** *N170 to happy expressions. The mean value was -3.6 µv before and -3.0 µv after the intervention. This difference is statistically significant.*

3.3.3. Waiting list control-group

In addition, the same analysis at the time points *pre* and *mid* was made to waiting list control-group. No significant interactions were found, only main effects for stimulus type (F(28.621)=22, p=0.001) and electrode site (F(4.960)=22, p=0.036).

3.3.4. The effects of intervention to combined groups

Added to this, the data was analyzed from different point of view by eliminating the effect of group. Thus, the experimental group and waiting list control-group were combined (n=42) and only the treatment effects were examined after every depressed subject had received the intervention. Almost significant four-sided interaction was found in this analysis, stimulus type × expression × electrode site × time of measurement; F(3.787)=41, p=0.059. Time of measurement had also almost significant main effect, F(3.698)=41, p=0.061. Stimulus type × expression × electrode site showed a significant effect, F(5.070)=41, p=0.030. Post hoc comparisons revealed

a significant effect in *fearful* at P7, t(-2.280)=41, p=0.028. *Happy* at P7 and P8 and *fearful* at P8 did not fulfilled the criteria of significance (p>0.05). The differential responses to fearful expressions diminished. The figures 8 and 9 present the N170 to happy and fearful expressions before and after intervention. As can be seen, the differences were not so large.

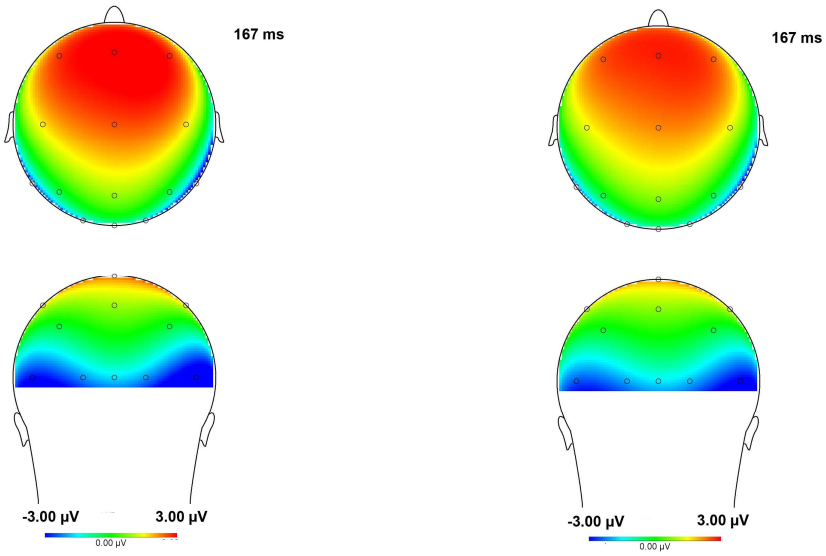

Figure 8. ***Both depressed groups (n=42).*** *N170 to happy expressions before and after the intervention.*

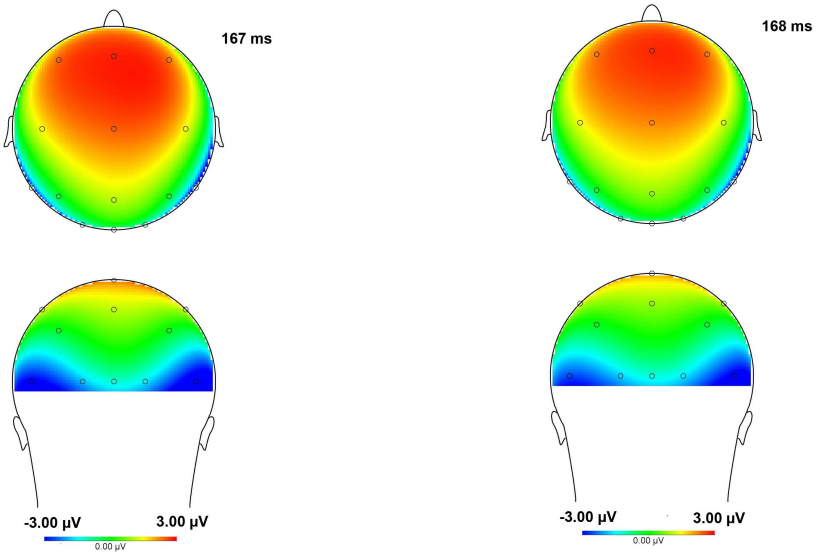

Figure 9. ***Both depressed groups.*** *N170 to fearful expressions before and after the intervention.*

The medication was used as a covariant in ANOVA analysis and it did not affect to ERP responses. Furthermore, there were no correlations between ERP amplitudes and age. When both experimental and waiting list control group have been treated, a changes in BDI scores and ERP responses (N170) were calculated. The correlation of these values (BDI and ERPs) were checked, but no correlations were found.

4. DISCUSSION

The aim of this study was to investigate the existence of face specific event-related potential N170 and change detection (vMMN) in the context of face perception among depressed and non-depressed subjects. In addition, the effects of short therapeutic intervention to brain functioning were in focus.

It appeared that the visual mismatch negativity is a phenomenon which elicits in both depressed and non-depressed subjects, although being different in the two groups. The responses elicited in different locations; the right hemisphere were dominant in non-depressed group while in depressed group the responses elicited mostly bilaterally. Therefore, visual mismatch negativity, a.k.a. change detection, existed in both groups. Depression did not effect on its appearance. The brain responses differed depending on electrode's localization, that is to say brain hemispheres: in non-depressed subjects the responses were larger in the right hemisphere areas whereas in depressed there were no big distinctions between hemispheres. An important finding in this study was that happy expressions did not elicit a significant response in left hemisphere, only in right hemisphere in non-depressed individuals. This finding is in line with previous studies which note that the elicitation of N170 is more remarkable in the right hemisphere in healthy subjects (Bentin et al., 1996; Itier & Taylor, 2004; Pazo-Alvarez, 2003; Fu et al., 2003). It became evident that happy facial expressions were lateralized to right while fearful expressions elicited responses bilaterally in non-depressed subjects. Results are in agreement with a finding whereby diminished activity of right hemisphere can be a risk factor for major depression (Heller & Nitschke, 1998; Bruder, 1995). It could be asked if there is a connecting factor between this discovery and processing happy expressions bilaterally, which the case was in depressed group.

The short therapeutical intervention with the means of acceptance and commitment therapy evoked some changes in brain functioning in depressed subjects. However, the differences between groups' baselines should be taken into consideration. There is evidence from previous studies, which are introduced by Roffman et al. (2005) in their review article, that psychotherapy has an impact on brain functioning. Aforementioned studies used functional neuroimaging. What is noteworthy, is an effect between experimental and waiting list control group regarding changes among cognitions.

Some changes occurred in response's localization, change detection and processing low-level features in faces in the course of time. The interaction still raises questions about the effects of intervention. However, as Roffman et al. (2005) note in their article, that it is uncertain whether the effects are caused by therapy or other factors. The mechanisms are still staying vague. The responses of the subjects in waiting list control group did not change when they were not having therapy, which gives support on our hypothesis. Meanwhile, responses in experimental group changed after therapy. What is noteworthy, is that responses only to happy faces changed remarkably and changes to happy expressions were larger than to fearful expressions. Therefore, some changes in processing low-level features in especially happy facial expressions have occurred. Why are the responses diminished? One interpretation is associated with habituation due to pre-post measurement: the familiar stimuli are eliciting smaller responses. Psychological effects can also be one concern. It is not clear what kind of processes happy and fearful expressions generate in brains and how the intervention influences to these processes. For example, can the therapy contribute to feel especially positive emotions and if so, how? Boyhuys et al. (1996) have pointed out, that hyposensitivity to negative facial stimuli can maintain depression. One assumption might be that therapy has trained subject's skills in dealing with emotions, for example, and affects to brain's event-related potentials as well – as diminished responses. It is also suggested that depressed individuals have a systematic cognitive bias in their information processing, which leads them to notice negative life events in more detail and ignore the positive interpretations (Beck, 2008; Mineka & Sutton, 1992; Shestuyk et al., 2005). However, in the light of these findings, our results related to different facial expressions seems quite ambiguous.

When the influence of time was ignored and the treatment effects were studied within all depressed subjects, it was found out that responses to fearful expressions in the left hemisphere diminished. The changes occurred in change detection, not in processing low-level features in faces as such. Though the result did not fulfill the statistical significance, it exhibits the underlying effects. What remains unclear, is that why significant effects are generated to fearful expressions in left hemisphere in this case. Because the results are not congruent with each other, these aspects require further study. Left hemisphere activity relates to anxiety and depressive mood, state Heller & Nitschke (1998) and Kayser et al. (2000).

There is evidence that preattentive visual processing is disturbed in other clinical states than in major depression as well, like in schizophrenia, mild cognitive impairment and Alzheimer's disease. Urban et al. (2008) points out that vMMN can work as a marker of progression in schizophrenia; schizophrenics also have systematically smaller vMMN responses than healthy subjects. In MCI and in Alzheimer's disease vMMN elicits abnormally (Tales et al., 2008). The following question could be, can vMMN serve as a precursor in different clinical states. Although (visual) mismatch negativity relates to cognition and it is fundamental quality in human beings, its significance and applications are not certain (Urban et al., 2008). Because the possible applications are not conceived yet, this field of study should investigate more closely in future.

Considering the suggestive results, it is evident that there are some limitations in the research frame. Because of the findings of previous studies of EEG and face perception (Itier & Taylor, 2004), we chose only two electrodes (P7, P8) to analyses although there would have been many other options (e.g. electrodes at occipital area, Tales et al., 1999 and at frontal area, Astikainen & Hietanen, 2009). In future, it would be interesting to investigate more closely the locations of event-related potentials. What are not studied here are the latencies of N170, which can also be revealing. Besides, on top of component N170, there are components P1, VPP and N200, for example, which have been used in studies related to face processing (Itier & Taylor, 2004; Deldin et al., 2000, Bentin et al., 1996). However, the choice we made proved to be promising because N170 seemed to be elicited in the inspected areas. Additionally, the chosen time window seemed to be suitable, as the previous studies also suggest (e.g. Astikainen & Hietanen, 2009; Pazo-Alvarez, 2003). The paradigm was well planned and successfully realized. The subjects were divided into groups which ensured most of the intervening factors. The size of the data was adequate for study that combines behavioral and EEG analyses. Nonetheless, the groups were very heterogeneous. The majority of subjects were women, which can be regarded as a limitation. In addition, some of the depressed subjects were medicated which can have an impact on brain's event-related potentials. The severeness of depression also varied between subjects. It also has to bear in mind that length and cause of depression were very different depending on individual. Reactive versus long-lengthy depression have presumably different underlying mechanisms which can explain the deviations in ERP responses. Additionally, the succeeding of the therapeutic intervention was varying.

Other subjects were more receptive to the therapy than other. In addition, the long-term effects of the therapy should be taken into closer consideration. It can also be asked, if the differences between ERPs were related to group baseline situation, not to the intervention at all. All things considered, psychological research will always have factors that can not be fully controlled.

What is noticeable, it was found out that vMMN elicits among depressed subjects, too. The results also confirm the findings from the lateralization of these event-related potentials – the right hemisphere dominance in healthy subjects (Bentin et al., 1996; Itier & Taylor, 2004; Pazo-Alvarez, 2003; Fu et al., 2003). Although the phenomenon exists in both depressed and non-depressed, the responses in healthy subjects are considerably larger and localizes mainly on right in processing happy expressions. Depressed subjects did not show as clear lateralization as the non-depressed subjects. It can be noted that human brain processes faces normally with the right hemisphere (Bentin et al., 1996) and this process is disturbed in the major depression (Kayser et al., 2000). On the grounds of these findings, it can be assume that depression affects in preattentive visual perception and information processing in some ways, at least in the context of facial expressions (Kayser et al., 2000; Gotlib et al., 1998). The negatively biased cognitive schemas (Beck, 2008) might have a connection to preattentive information processing.

The treatment effects proved that short therapeutical intervention can contribute both to self-assessed mood and in preattentive visual information processing in the case of depression. This supports finding that psychological intervention can have an influence on brain functioning and that way on mood and behaviour.

5. CONCLUSION

Although the results were partly quite puzzling, preattentional information processing and the use of event-related potentials can reveal new findings from facial expressions recognition and major depression. The findings of this study illustrated that there are some crucial variation in visual information processing between depressed and non-depressed. This study also showed that even a short therapeutical intervention can cause changes in brain functioning. These findings are interesting and need closer examination in future.

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