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**Facial electromyography in anxious and nonanxious depression:
Relation to auditory perceptual asymmetry and measures of
experienced emotion**

Voglmaier, Martina Maria, Ph.D.

City University of New York, 1991

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**FACIAL ELECTROMYOGRAPHY IN ANXIOUS AND NONANXIOUS
DEPRESSION: RELATION TO AUDITORY PERCEPTUAL
ASYMMETRY AND MEASURES OF EXPERIENCED EMOTION**

by

Martina M. Voglmaier

**A dissertation submitted to the Graduate Faculty
in Psychology in partial fulfillment of the
requirements for the degree of Doctor of
Philosophy, The City University of New York**

1991

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Abstract

FACIAL ELECTROMYOGRAPHY IN ANXIOUS AND NONANXIOUS
DEPRESSION: RELATION TO AUDITORY PERCEPTUAL
ASYMMETRY AND MEASURES OF EXPERIENCED EMOTION

by

Martina M. Voglmaier

Advisor: Professor Gad Hakerem

Facial electromyography (EMG) in response to pictures of facial affect and dichotic listening measures of perceptual asymmetry were assessed in anxious- and nonanxious-depressed patients and normal controls. All subjects were female and most patients met criteria for an "atypical" depression. Anxious-depressed patients also met criteria for an anxiety disorder, primarily panic disorder. Anxious-depressed patients showed a striking lack of facial muscle reactivity to happy faces, a lack of normal right ear advantage (REA) on a verbal dichotic listening task, and a lack of normal association between facial muscle activity and self-reported happiness in response to happy faces. In contrast, nonanxious-depressed patients showed more distinct corrugator muscle activity in response to sad faces, a slightly larger REA on a verbal dichotic task, and an abnormally strong association between corrugator activity and self-reported sadness in response to sad faces. All depressed patients showed a lack of normal left ear advantage (LEA) on a nonverbal dichotic task. Facial muscle activity was found to correspond to self-reported experience of emotion, but expression-experience associations varied

according to the affective quality of the facial stimuli, and differed for the depressive subtypes. Successful antidepressant treatment was found to reduce facial muscle activity levels in the nonanxious depressed group, but did not change patterns of facial muscle activity in response to pictures of affective faces. There was some preliminary evidence that differential patterns of corrugator muscle activity may characterize treatment responders and nonresponders. The results suggest that anxious atypical depression may be characterized by more left hemisphere involvement than nonanxious atypical depression, and support valence-lateralized hypotheses of emotion. It is suggested that abnormal facial muscle activity patterns in these depressive subtypes may be state-independent characteristics, and that abnormal expression-experience associations may be related to social deficits in depression.

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Introduction and Literature Review

A. Purpose of the Study

The neuropsychological literature suggests that the right hemisphere (RH) is specialized for both the expression and perception of facial emotion (see reviews in Borod & Koff, 1984; Borod, Koff, & Caron, 1983). Right hemisphere dysfunction has been implicated in major depressive disorder (e.g., Coffey, 1987; Flor-Henry, 1976; Otto, Yeo, & Dougher, 1987) and in panic disorder (e.g., Reiman et al., 1984; 1986). In light of these findings, the measurement of facial emotional expression in depressed individuals with and without anxiety disorders would appear to be an important avenue for neuropsychological investigation.

Several studies have assessed facial emotional responses in depressed patients (e.g., Carney, Hong, Brim, Plesons, & Clayton, 1983; Greden, Genero, Price, Feinberg, & Levine, 1986; Schwartz, Fair, Salt, Mandel, & Klerman, 1976a, 1976b). In general, findings from electromyographic (EMG) studies suggest alterations of characteristic "positive" and "negative" facial muscle activity patterns, which may be related to severity and subtype of depression, and may predict treatment response (see review in Fridlund & Izard, 1983). However, few studies have attempted to relate these findings to hypotheses of RH dysfunction in depression, or RH superiority for emotional processing. In addition, no studies have assessed "positive" vs. "negative"

facial emotional responses in depressed patients with anxiety disorders. Anxiety disorders are of interest on two grounds: a) lateralized hemisphere dysfunction has been suggested in anxiety (Tucker, 1981) and in panic disorder (e.g., Reiman et al., 1984); and b) widespread muscle activation characterizes anxious individuals (e.g., Fridlund, Hatfield, Cottam, & Fowler, 1986).

Given the hypotheses of right hemisphere involvement in facial emotion, and of right hemisphere dysfunction in both depressive disorder and panic disorder, one might predict that depressed patients with anxiety disorders would be particularly likely to show abnormalities of facial emotional expression. Although there have been previous studies of facial EMG in depressed subjects, none have examined the effects of comorbidity of anxiety and depression.

The purpose of the proposed study was to further study the role of the right hemisphere in facial emotion processing in depressed patients and, in particular, to contrast facial emotion processing in depressives with and without anxiety disorders. This was accomplished by employing objective facial affective stimuli to evoke "positive" and "negative" facial EMG patterns in anxious and nonanxious depressed patients and normal control subjects. In addition, given the hypotheses of right brain dysfunction in these disorders, this study related facial EMG measures

to dichotic listening measures of perceptual asymmetry. The relationship between facial EMG activity and experienced emotion was also measured by relating EMG data to self-report measures of immediate affective experience, to measures of depression, anxiety and anhedonia, and by examining changes in facial EMG following successful treatment of depression.

B. Psychophysiology and Facial Emotion

Historically, Darwin (1872) postulated that emotional processes are directly related to emotional expression, and that such expressions have a biological basis. Since then, facial expressions have been a focus for researchers of emotion. Several modern emotion theorists have incorporated this view of biologically programmed emotions (e.g., Izard, 1971, 1972, 1977; Tomkins, 1962). Most major theories agree that there are certain fundamental emotions, each with a distinct, innately programmed neurophysiological substrate, universally recognized facial expression, and motivational subjective experience (e.g., Izard's "Differential Emotions Theory"). The interest in facial expressions has focused not only on the function of the face as a primary organ for the expression and communication of emotions (Borod, Koff, & Caron, 1983), but also on the possible role of the facial muscles in supplying sensory feedback for the subjective experience of emotion, e.g., the "facial feedback" hypothesis (see Buck, 1980, for review).

Electromyography (EMG) has been used to investigate the physiological properties of differential facial emotions. The EMG signal is the electrical manifestation of muscle contraction, and has several advantages over other methods of measuring facial behavior (see Ekman, 1981, for review). First, the EMG signal is detected instantly and lends itself to immediate recording, whereas other coding systems require

large amounts of time for observer judgments and manual recording of facial actions. Second, the EMG signal is a very precise index of the degree of a muscle contraction, offering a more fine-grained measure of muscle activity than that provided by raters. Third, the use of EMG techniques allows the detection of muscle contractions which are too small to be observable, providing for detection of possible "covert" facial expressions (Fridlund & Izard, 1983). Finally, changes in facial muscle activity over time can be readily detected and quantified.

1. Normal Subjects

In EMG studies measuring covert facial muscle activity under various affective conditions, the most robust finding is that two facial muscles in particular, the corrugator and zygomatic (see Figure 1), appear to be effectors of patterns of "negative" and "positive" affect, respectively (see Fridlund & Izard, 1983). The characteristic "positive" muscle pattern consists of an increase in zygomatic muscle (which retracts the corners of the mouth into a smile) activity, and the "negative" pattern consists of an increase in corrugator muscle (which knits the brow into a frown) activity. Schwartz and his colleagues (Brown & Schwartz, 1980; Schwartz, Brown, & Ahern, 1980; Schwartz et al., 1976a, 1976b; Schwartz, Fair, Mandel, Salt, & Meiske, 1978) using affective imagery paradigms in which subjects are required to imagine certain affective scenes, have

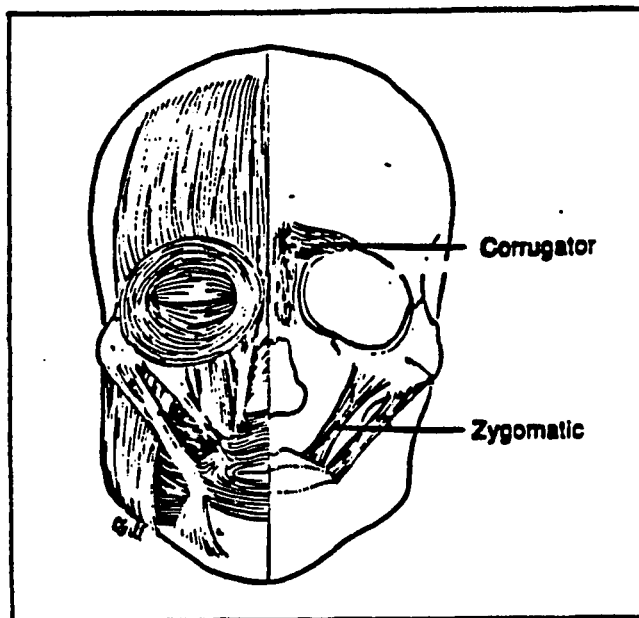


Figure 1. Anatomic illustration of facial musculature. [From: Greden, et al., 1986; reprinted with permission.]

consistently reported increases in zygomatic EMG activity during the imagination of happy and pleasant scenes, and increases in corrugator activity during the imagination of sad and unpleasant scenes. These characteristic facial muscle activity patterns have also been found in normal subjects during mood induction procedures (Sirota & Schwartz, 1982), and in response to affective self-referent statements (Sirota, Schwartz, & Kirsteller, 1987), reflective questions (Schwartz, Ahern, & Brown, 1979), and affective visual stimuli (Cacioppo, Petty, Losch, & Kim, 1986). In light of the usual functional roles of the corrugator and zygomatic muscles in overt facial display, these findings are generally interpreted to support a "differential emotions" viewpoint, in which distinct patterns of facial muscle activity are related to distinct emotions.

In a series of studies designed to measure the affective impact of social interaction, Dimberg (1982, 1987, 1988; see review, 1990) found that mere presentation of still photographs of posed facial expressions was sufficient to elicit characteristic facial EMG responses in normal subjects. Subjects showed increased corrugator activity in response to pictures of angry faces, and increased zygomatic activity in response to pictures of happy faces. In a later study, Dimberg (1987) had subjects rate the faces for "pleasantness" or "unpleasantness", and found that angry

faces were rated as being more unpleasant and happy faces were rated as being more pleasant. Dimberg (1988) also found that angry faces evoked increased corrugator activity, decreased heart rate, and more subjective experience of fear, while happy faces evoked increased zygomatic activity, and increased subjective experience of happiness. Dimberg (1989) has concluded that "the facial muscles constitute an emotional output system" and are "intimately associated with the experience of emotion". While Dimberg recognized the possibility that these facial reactions may be a form of "social mimicry", his findings that the same patterns were evident in response to "fear-relevant and -irrelevant" visual stimuli (e.g., snakes vs. flowers), would contraindicate an explanation of simple mimicking behavior (Dimberg, 1986, 1989). Similar results have been found in response to pictures of happy and sad faces (Voglmaier & Hakerem, 1988 [see Appendix A]; Wexler, 1989).

Other, less robust, findings include a differential, emotion-dependent time course of the corrugator and zygomatic muscles (Sirota & Schwartz, 1982; Sirota, Schwartz, & Kirsteller, 1987; Voglmaier & Hakerem, 1988), sex differences (Schwartz et al., 1980; Dimberg & Lundquist, 1988), and laterality differences (Schwartz, et al., 1979; Sirota & Schwartz, 1982). Laterality differences will be discussed in a later section of this review.

In summary, the results of facial EMG studies in normal

subjects suggest that EMG is a sensitive indicator of covert muscle activity, that activity in the corrugator and zygomatic muscles can differentiate "negative" and "positive" affective stimuli, and that activity in these muscles may be related to emotional experience. Because pictures of facial affect represent a more objective method for evoking facial muscle responses than other methods (e.g., imagery, questions), they were used to elicit facial activity patterns in the current study.

2. Depressed Subjects

Most studies which measure expressive facial emotion in depressed patients have investigated spontaneous facial muscle activity using EMG measures. Some have been related to theories of "differential" emotions and the "facial feedback" hypothesis. Others have attempted to employ EMG as a physiological marker for depression, subtypes of depression, or treatment response. The following is a review of the literature.

Schwartz et al. (1976a) were the first to measure facial electromyography in depressed individuals, and employed the method of affective imagery to evoke spontaneous facial muscle activity. Schwartz et al. asked subjects to think about situations which made them happy or sad, and then asked them to imagine going through a typical day. In control subjects, corrugator muscle activity was increased during the "sad" condition, and decreased during

the "happy" condition, the "characteristic" facial muscle pattern. In depressed subjects, corrugator muscle activity did not show the normal decrease during the happy condition (in fact, a slight increase was found). The greatest difference between the groups was seen during the "typical day" imagery. While control subjects showed a decrease in corrugator activity, depressed subjects showed an increase in corrugator activity. These researchers hypothesized that the increased corrugator activity in depressed patients was evidence of a "miniature sad facial expression" during neutral and happy stimuli. In a related study, Schwartz et al. (1976b) found that when asked to "think" about affective scenes, depressed patients showed less zygomatic increase in response to happy imagery, and a greater corrugator increase in response to sad imagery compared to controls. However, when asked to "feel" the emotion of the imagery, facial EMG magnitudes were near normal levels. Schwartz et al. suggested that depressed patients were not spontaneously generating muscle patterns for the feedback necessary to experience emotion, but could do so when given explicit instructions, which these researchers suggested might have implications for cognitive therapy. Brown (1982) showed a greater association between expressed and experienced mood states (measured by facial EMG and subjective ratings, respectively) during happy imagery in normals, and a greater association during sad imagery for depressed subjects. She

suggested that depressed individuals may not have the appropriate screening mechanisms necessary to protect them from negative or painful stimuli, and suggested that the use of biofeedback in therapy might help patients work toward more normal associations of emotional expression and experience.

Greden, Genero, and Price (1985), using drug-free right-handed females, found that subjects who met criteria for major depressive disorder showed increased corrugator activity during rest, happy, and "typical day" imagery when compared to controls. A significant correlation was also found between corrugator activity and the "agitation" subscore on the Hamilton Depression Scale, perhaps implicating anxiety in elevations of facial muscle magnitude. Greden et al. (1986) examined facial muscle activity in endogenous and nonendogenous subtypes of depressed patients during affective imagery. In general, depressed patients did not show the distinct patterns of muscle activity evidenced by controls, but only the endogenous subtype differed significantly from normal control subjects in facial EMG magnitude. Endogenous patients showed increased zygomatic activity at rest and in response to sad and "typical day" imagery, and increased corrugator activity in response to sad imagery. Nonendogenous depressed patients did not differ from normal controls in facial EMG magnitude. Compared to nonendogenous

patients, endogenous depressives showed increased corrugator activity during the happy imagery condition. Similar results were recently obtained by Wexler (1989), who examined zygomatic and corrugator muscle activity in patients with major depression in response to pictures of facial affect. In comparison to control subjects, depressed patients showed increased corrugator and zygomatic activity and, as in Greden et al.'s (1986) study, failed to show the emotion-specific patterns of activation seen in control subjects. The results of these studies may implicate a lack of "emotional reactivity" in depression.

Studies of resting facial muscle activity generally show increased EMG magnitude in depressed patients. Carney, Hong, Kulkarni, and Kapila (1981a), measuring left-side corrugator activity and skin conductance levels, found that female depressed patients showed increased corrugator activity at rest in comparison to controls, and that more severely depressed patients had decreased skin conductance levels (SCL). Scores of self-rated depression on the Beck Depression Inventory (BDI) were not associated with EMG or SCL levels. Carney et al. concluded that only increased corrugator activity differentiates depressed from nondepressed subjects at rest, perhaps implicating somatic nervous system overarousal. Carney et al. (1983) found that "reactivity" of depression was associated with increased corrugator activity. "Reactivity" in this study was measured

following review of patients' hospital charts for evidence of psychomotor activity and responsivity to social interaction.

Several studies have associated facial muscle activity in depressed patients with treatment response. Schwartz, et al. (1978) found that increased resting corrugator activity in depressed patients prior to treatment predicted improvement after 2 weeks of treatment with either a tricyclic antidepressant (TCA) or placebo. This was true for improvement as indexed by the clinician-rated Hamilton Depression Scale, but not the self-rated Zung scale. This improvement in depressive symptoms was associated with a reduction in corrugator activity at retest. The authors suggested that the depressed state is accompanied by a "miniature sad facial expression" at rest, which diminishes following alleviation of depression. Carney, Hong, O'Connell, and Amado (1981b) found that both increased zygomatic and corrugator activity prior to TCA treatment predicted improvement after 2 weeks, as measured by the self-rated BDI. However, there was no reduction in corrugator activity associated with the reduced BDI scores. Greden, Price, Genero, Feinberg, and Levine (1984), studying drug-free endogenously depressed subjects, found that increased zygomatic activity predicted response to TCAs.

Conclusions regarding the facial emotion of depressed patients from EMG studies remain tentative, due to the small

number of studies, inconsistent findings, and methodological problems in a number of studies. Patients have not always been diagnosed by strict diagnostic criteria, and in those studies assessing the association between EMG activity and treatment response, patients have not always been tested drug-free prior to treatment, or after adequate time periods on antidepressant medications. In addition, the effect of anxiety levels or presence of anxiety disorders in depressed patients has not been assessed. Anxiety levels are of importance in studies of facial EMG, because widespread muscle activation has been found to characterize anxious individuals (e.g., Fridlund et al., 1986). The most robust findings from strictly diagnosed depressed patients have been: a) increased levels of EMG activity (either corrugator or zygomatic) in comparison to control subjects, which may be specific to subtype and may predict treatment response, and b) a failure to show the distinct facial muscle pattern of normal control subjects in response to affective stimuli. In the present dissertation, patients were diagnosed according to DSM-IIIr (Diagnostic and Statistical Manual of Mental Disorders, 3rd edition, revised; American Psychiatric Association, 1987) criteria, were initially tested drug-free, and were retested after at least 6 weeks of antidepressant treatment. The presence of anxiety disorders and levels of anxiety and depression were accounted for by contrasting anxious and nonanxious depressives, and

employing self-report measures of "state" and "trait" anxiety and depression. Diagnosis and treatment response were assessed by research psychiatrists.

C. Neuropsychology and Facial Emotion

Emotion research has developed beyond the physiological level, to include theories of lateralized brain dominance. Clinical and experimental studies in normal, brain damaged, and psychiatric patients suggest that the right hemisphere may be uniquely specialized for the perception, experience, and expression of emotion (e.g., Coffey, 1987; Joseph, 1988; Ley & Bryden, 1982; Tucker, 1981). The bulk of neuropsychological studies of facial emotion employ methods to measure asymmetries of both the expression and perception (or appreciation) of facial emotion, and have provided evidence for right hemisphere superiority in the processing of facial emotion (see Borod & Koff, 1984; Borod, Koff, & Caron, 1983 for reviews). The following review will include the major findings in both normal and brain-damaged subjects which suggest right hemisphere superiority for the expression and appreciation of facial emotion.

1. Facial Expression of Emotion

Before beginning a review of lateralized facial expression, the proposed neuroanatomical pathways which mediate posed and voluntary facial expression, and pathways which innervate portions of the face must be understood. There is evidence from brain damaged individuals that

voluntary and spontaneous expressions of emotion are mediated by different pathways (see Rinn, 1984). Voluntary facial expressions are believed to be mediated by a pathway which extends from the motor cortices to the seventh cranial (or "facial") nerve nuclei in the brain stem. The muscles of the upper face (forehead and upper eyelid) are subserved by the ventral portions of the nuclei, and are believed to be under bilateral control. The muscles of the lower two-thirds of the face (the lower eyelids, and the nose, lips, cheeks, and neck) are subserved by the dorsal portions of the nuclei, and are believed to be contralaterally innervated.

The neuroanatomical pathways mediating spontaneous, involuntary expressions of emotion are believed to originate in the thalamus or globus pallidus, involve portions of the limbic system, and follow extrapyramidal pathways. Based on evidence that both subcortical and cortical lesions may produce emotional outbursts, the involuntary expression of emotion is believed to be mediated both by subcortical and cortical centers, with bilateral and possible contralateral innervation of the face (see Etcoff, 1986).

a. Normal Subjects. Because the lower portions of the face are thought to be contralaterally innervated (Kuypers, 1958; DeJong, 1978), measurement of asymmetries of posed facial expression are used to study the neuropsychological mechanisms underlying the facial expression of emotion. This is usually achieved by subjective ratings or measurement of

muscle displacement of the two sides of the face, or by rating the intensity of mirror-image hemiface composites. Most consistently, in right handed adults, posed facial expressions are found to be more intense on the left side of the face (e.g., Borod & Caron, 1980; Dopson, Beckwith, Tucker, & Bullard-Bates, 1984; Ekman, Hager, & Friesen, 1981; Heller & Levy, 1981; Sackeim, Gur, & Saucy, 1978). This asymmetry is thought to reflect right hemisphere dominance for posed facial expression. However, there is some evidence that negative facial expressions may be mediated by the right hemisphere, while positive facial expressions may be mediated bilaterally or by the left hemisphere (e.g., Borod, Koff, & White, 1983 in females; Sackeim & Gur, 1978).

The evidence for lateralization of spontaneous emotion is inconclusive. Some studies have found evidence for right hemisphere lateralization (Dopson et al., 1984; Moscovitch & Olds, 1982), others for no lateralization (Ekman et al., 1981), and still others for valence-dependent lateralization of emotional facial expression (Borod et al., 1983). Interestingly, in studies in which both spontaneous and posed facial expressions are measured in the same subjects, no differences in lateralization have been found (Borod, Koff, & White, 1983; Dopson et al., 1984). Although the validity of lateralized EMG measures has been questioned (Fridlund, 1988), Schwartz et al. (1979) found differential

laterality effects for the zygomatic muscle, depending on the emotional valence of "reflective" questions. Involuntary zygomatic activity was increased on the right relative to the left side of the face during "positive" affective questions, and the reverse was true for "negative" affective questions. The corrugator response was lateralized (increased on the left) only during voluntary facial expressions. Sirota and Schwartz (1982) found similar lateralization effects for self-referent mood statements.

In summary, the literature suggests that in posed facial expressions, the left half of the face is more expressive than the right half of the face, perhaps particularly for negative facial expressions. This is consistent with hypotheses of right hemisphere specialization for facial expression, although there is some evidence that positive facial expressions may be mediated bilaterally or by the left hemisphere. The evidence for lateralization of spontaneous expressions is inconclusive.

b. Brain Damaged Subjects. There are few studies of facial expression in brain damaged individuals. Both Buck and Duffy (1980), who studied spontaneous facial expressions, and Borod, Koff, Perlman-Lorch, and Nicholas (1986), who studied both posed and spontaneous facial expressions, found patients with right unilateral brain damage to be less facially expressive than left brain damaged subjects and controls. Ross and Mesulam (1979),

reported infrequent facial emotional responses in two right-brain damaged patients. In contrast, Kolb and Milner (1981) found no differences between facial expressiveness of right and left brain damaged patients. Recently, Kent, Borod, Koff, Welkowitz, and Alpert (1990) found that right brain damaged individuals showed significant deficits in the accuracy with which they posed positive (but not negative) facial emotions compared to left brain damaged patients and controls.

In summary, the few studies of facial expression in brain damaged patients suggest that those with right hemisphere pathology may be impaired in facial emotional expression, consistent with hypotheses of right hemisphere specialization for facial expression of emotion. There is recent evidence that these deficits may be specific to positive facial emotions during posed facial expression.

2. Appreciation of Facial Emotion

Before reviewing studies which examine the perception of facial emotion in both normal and brain-damaged populations, a review of specific brain areas which appear to mediate the perception of faces and facial expressions is necessary. There is evidence from neural cell recording in nonhuman primates, that the perception of faces is mediated by regions that lie in the right occipital-temporal lobe (Perrett, Rolls, & Caan, 1979, 1982; Rolls, 1981). Cells in the fundus of the superior temporal sulcus of the RH have

been found to respond preferentially to faces, and to different regions of the face. Similarly, in humans, the areas most thought to mediate the perception of faces include the right anterior inferior occipital region of the occipital temporal junction (Meadows, 1974), the ventromedial sector of the occipitotemporal cortex, the white matter of the posteroinferior temporal lobes and the inferior occipital lobes (Damasio, Damasio, & Van Hoesen, 1982).

Similar brain areas have been cited in the perception of facial expressions in particular. From studies of both normal and brain damaged adults, the right occipital-temporal lobe appears to be important in the perception and recognition of facial expressions (Nelson, 1987). The following is a review of the literature.

a. Normal Subjects. Neuropsychological studies of the perception of facial emotion typically involves divided visual field presentation of affective faces. In these studies, the right hemisphere has been repeatedly found to be faster and more accurate in recognizing facial expressions of emotion than the left hemisphere (e.g., Ley & Bryden, 1979; Ladavas, Umiltà, & Ricci-Bitti, 1980; Strauss & Moscovitch, 1981). In addition, when chimeric faces (e.g., faces which show different emotional expressions in each half-face) are presented to normal subjects, the expression in the subject's left visual field has been found to have

the most impact (Campbell, 1979; Heller & Levy, 1981). This specialization for perceiving facial emotion has been found to be independent of the right hemisphere's specialization for recognizing faces (Suberi & McKeever, 1977; Ley & Bryden, 1979), and for visuospatial perception (McKeever & Dixon, 1980) in general.

As in studies of facial expression of emotion, there is some evidence that negative emotional expressions may be better perceived by the right hemisphere, while positive expressions are better perceived by the left hemisphere (Reuter-Lorenz & Davidson, 1981). However, the bulk of the literature supports a right hemisphere specialization for the perception of facial emotion, regardless of affective valence.

b. Brain Damaged Subjects. In clinical studies, patients with right unilateral brain damage have been found to show greater impairment in the recognition of facial expressions of emotion than those with left brain damage (e.g., Borod, Koff, Perlman-Lorch, & Nicholas, 1986; Cicone, Wapner, & Gardner, 1980; Benowitz, Bear, Mesulam, Rosenthal, Zaidel, & Sperry, 1983; Etcoff, 1984a, 1984b). DeKosky, Heilman, Bowers, and Valenstein (1980) found that right brain-damaged patients had deficits in naming facial emotions as well as in discriminating photographs of neutral vs. emotional expressions in photographs of the face. Recently, Rapcsak, Kaszniak, and Rubens (1989) described a

patient with right temporal lobe damage with a selective impairment in naming and pointing to emotional facial expressions. In addition, recognition of facial emotion has been found to be disrupted by stimulation of the right middle temporal gyrus in patients undergoing surgery for intractible epilepsy (Fried, Mateer, Ojemann, Wohns, & Fedio, 1982).

In summary, the majority of neuropsychological studies in both normal and brain-damaged individuals generally support a right hemisphere specialization for the expression and perception (appreciation) of facial emotion.

D. Cerebral Laterality in Emotional Disorders

Research on the neuropsychology of emotional disorders has indicated that particular psychiatric disorders may be associated with abnormal hemispheric asymmetries (see Tucker, 1981; Wexler, 1980). The following review will focus on the major findings suggesting lateralized brain involvement in depression and anxiety.

1. Depression

There is growing evidence suggesting right hemisphere involvement in depression (see Flor-Henry, 1979; Otto et al., 1987; Tucker, 1981; Wexler, 1980, for reviews). Depressive reactions have been associated with left brain damage (Robinson, Kubos, Starr, Rao, & Price, 1984) and left hemisphere inactivation by sodium amytal injection (Gainotti, 1972), both of which could result in relative

right hemisphere activation. Relatively greater activation of the right frontal region, as measured by EEG alpha activity, has been found in depressed psychiatric patients (Perris, Monkhorst, Von Knorring, Botskarev, & Nikiforov, 1978; Schaffer, Davidson, & Saron, 1983), and in normal control subjects following depressed mood-induction (Tucker et al., 1981) and during negative emotion-evoking questions (Ahern & Schwartz, 1985).

Additionally, depressed patients have been found to show poor performance on neuropsychological tasks of right hemisphere function (e.g., perceptual tasks using visuospatial stimuli; see Goldstein, Filskov, Weaver, & Ives, 1977), which may improve with alleviation of depression (Kronfol, deS. Hamsher, Digre, & Waziri, 1978). Studies using dichotic listening tasks have shown evidence of abnormal perceptual asymmetry in depressed patients (see Bruder, 1988, for review). Although the auditory system contains both ipsilateral and contralateral pathways from the inner ear to auditory cortex in the temporal lobe, under conditions of dichotic listening (i.e., simultaneous stereo presentation of different stimuli to each ear), the ipsilateral connections are thought to be suppressed so that auditory perception is primarily contralateral (Kimura, 1967). In right-handed normal subjects, a relative right ear advantage (REA) for verbal material is thought to reflect left hemisphere dominance for language, while a left ear

advantage (LEA) for nonverbal material is thought to reflect right hemisphere dominance for nonverbal material. In depressed subjects, evidence for right hemisphere dysfunction has been most consistently seen for nonverbal dichotic tasks (musical tones, clicks), where depressed patients fail to show the LEA seen for control groups (Berger-Gross, Bruder, Quitkin, & Goetz, 1985; Bruder, Sutton, Berger-Gross, Quitkin, & Davies, 1981; Johnson & Crockett, 1982; Yozawitz, et al., 1979). Abnormal findings using verbal dichotic tasks (e.g., a larger than normal REA) have been less consistent (e.g., Colbourn & Lishman, 1979; Wexler & Heninger, 1979), and may be related to diagnostic subtype (e.g., Bruder, Quitkin, Stewart, Martin, Voglmaier, & Harrison, 1989, Wale & Carr, 1990). Several studies have found that unipolar depressed patients fail to show a normal REA on verbal tasks, which may be related to severity of depression (Moscovitch, Strauss, & Olds, 1981; Johnson & Crockett, 1982). This has been variously explained as a decline in left hemisphere function, over-priming of the right hemisphere, or shifts in relative activity of each hemisphere.

While the majority of studies supporting unilateral hemisphere dysfunction in depression implicate right hemisphere involvement, there is some evidence to implicate left hemisphere dysfunction in depression. Left frontal hypoactivation on EEG has been found in depressed patients

(Henriques & Davidson, in press), and normothymic patients with a history of major depression (Henriques & Davidson, 1990). Davidson (1984, 1987) has suggested that the basis for frontal EEG asymmetries is approach and withdrawal, with the left anterior brain region specialized for approach behaviors, and the right anterior region specialized for withdrawal behaviors. He views decreased left anterior EEG activation as a predisposition to emotions associated with deficits in approach (e.g., sadness and depression). He suggests that individuals with accentuated right anterior EEG are more vulnerable to withdrawal-related emotions (e.g., fear, disgust, anxiety disorders) [Davidson & Tomarken, 1989].

a. Facial Emotion Processing in Depression. In light of the findings suggesting right hemisphere dysfunction in depression, and evidence for right hemisphere superiority for processing facial emotion, deficits in the expression and/or perception of facial emotion would be expected in depressed individuals. However, surprisingly few studies have attempted to relate facial emotional processes in depression to right hemisphere function. Jaeger, Borod, and Peselow (1986), using an observer-rated facial coding system, found that unipolar depressed patients were impaired with respect to the accuracy and intensity of their posed emotional expressions, particularly for positive expressions of happiness and surprise. Jaeger et al. related these

findings to a selective impairment for positive relative to negative emotion found in depressed patients for both prosodic output (Levin, Hall, Knight, & Alpert, 1985) and affective responses to facial stimuli (Schaffer et al., 1984). Similarly, Voglmaier and Hakerem (1988; Appendix A) found that in nondepressed control subjects, high scores on a "trait" depression scale were related to reduced zygomatic EMG activity in response to pictures of happy faces. This selective impairment in the expression of positive relative to negative emotion may be related to anhedonia (i.e., a reduced ability to experience pleasure), and may represent a state-independent characteristic.

Several studies have found depressed subjects to show abnormalities in the appreciation of facial emotion. Jaeger et al. (1987) examined the performance of depressed males on an emotional chimeric faces task, an emotion appreciation task which usually yields a left hemispace bias in normal control subjects (Levy, Heller, Banich, & Burton, 1983). Unipolar depressed patients were less lateralized (i.e., showed a smaller left hemispace bias) than normals in the perception of emotional chimeric faces, consistent with the literature showing atypical patterns of lateralization among unipolar depressives on tasks involving right hemisphere functions. Anxious-depressed children have been found to be less accurate than normal children in recognizing facial expressions of emotion (Walker, 1981), and depressed adults

were found to be less accurate than controls at labeling facial emotions, although they performed normally on a nonverbal facial emotion discrimination task (Feinberg, Rifkin, Schaffer, & Walker, 1986). Finally, Cooley and Nowicki (1989), using a reaction-time paradigm, found that depressed subjects were slower than normal controls in discriminating facial expressions of emotion. These researchers suggested that because of their slow processing of facial affect, depressed persons may fail to decode and thus misinterpret rapidly changing social cues, which may be related to the interpersonal and social skill deficits often present in depression.

The major psychosocial theories of depression consider social deficits to be crucial for the onset and/or perpetuation of depression. Lewinsohn's (1974, 1975) theory focuses on emitted social behaviors. He postulates that depression is caused by a low rate of response-contingent positive reinforcement, and has found that depressed persons have a lower activity level and lower number of pleasant activities than nondepressed people (e.g., Lewinsohn & Libet, 1972; MacPhillamy & Lewinsohn, 1974). The poor social skills of the depressed person are thought to contribute to this reduction in response-contingent positive reinforcement. Coyne's (1976a; 1976b) theory focuses on the reactions of other people to the depressed person, and assumes that the person accurately perceives negative social

information and that his or her behavior creates interpersonal negativity and rejection. Several studies have found that normal subjects feel or display negative reactions when interacting with a depressed person (e.g., Howes & Hokanson, 1979; Strack & Coyne, 1983). Finally, Beck (1963; 1967; 1971) emphasizes cognitive distortion of experience, which result in a negative conception of the self, world, and future. The negative conceptions result from dysfunctional schemas developed in early life experience which lead to systematic errors in thinking.

In summary, the few studies of facial emotion processing in depression support abnormalities in both the expression and the appreciation of facial emotion. These findings support the notion of right hemisphere processing abnormalities in depression. There is some suggestion that these abnormalities may be related to social deficits in depression.

b. Subtypes of Depression. Unipolar depression appears to consist of several subtypes (e.g., endogenous vs. nonendogenous). Establishing the validity of subtypes is important in improving treatment and understanding etiology. One subtyping dichotomy that appears promising for identifying drug-responsive groups is the "atypical" vs. "melancholic" distinction. Atypical depression represents a subset which is thought to respond preferentially to monoamine oxidase inhibitors (MAOIs), while endogenous and

melancholic depressions respond well to tricyclic antidepressants (TCAs) [Liebowitz et al., 1984]. Atypical depression is characterized by atypical symptom features, such as overeating, oversleeping, prominent anxiety, reactivity of mood, rejection sensitivity, extreme fatigue, and bodily inertia (leaden paralysis). Melancholia involves the same features as endogenous depression (e.g., anorexia and hyposomnia), but patients also exhibit pervasive anhedonia and nonreactivity of mood to usually pleasurable stimuli. Atypical depressives have also been shown to be distinguishable from endogenous depressives on the basis of their sleep characteristics (Quitkin et al., 1985).

The melancholia/atypical subtyping distinction also appears to be promising from a neuropsychological viewpoint. Recently, Bruder, et al. (1989) found that depressed patients with melancholic features, but not those with "atypical" features, showed abnormalities of perceptual asymmetries on both a verbal and nonverbal dichotic listening task. The abnormalities shown by melancholic patients were consistent with right hemisphere dysfunction (e.g., they showed an abnormally large right ear advantage on the verbal task, and failed to show the expected left ear advantage on a nonverbal task). Furthermore, Bruder et al. (1990) found that, in depressed patients, failure to show a right hemisphere (left ear) advantage on a complex tone task was associated with favorable response to tricyclic

antidepressant treatment. Poor left ear performance on the same complex tone task has also been associated with high measures of trait depression in non-depressed control subjects (Bruder & Voglmaier, 1990). Because poor performance in discriminating complex tones may implicate primary auditory cortex and surrounding regions of the right temporal lobe (Zatorre, 1988), the aforementioned studies suggest a link between some depressions and right hemisphere dysfunction.

The use of these verbal (speech discrimination) and nonverbal (complex tone discrimination) dichotic listening tasks to provide converging evidence of unilateral hemisphere (especially temporo-parietal) dysfunction is supported by studies of both normal and neurological populations. The strong association between language and the left hemisphere in most of the population (e.g., Rasmussen & Milner, 1977) suggests that the properties of speech perception provide at least an approximate description of cortical auditory function in the left hemisphere (e.g., Studdert-Kennedy & Shankweiler, 1970; Oscar-Berman, Zurif & Blumstein, 1975; Schwartz & Tallal, 1980). An analogous functional association for the right auditory cortex appears to be complex pitch perception (Sidtis, 1981), which has been found to be disrupted in patients with right (compared to left) middle cerebral artery (MCA) stroke (Sidtis & Volpe, 1988). When verbal (consonant-vowel) and nonverbal

(complex pitch perception) tasks were presented dichotically to patients with infarctions of the left or right MCA, the left hemisphere-lesioned group was impaired in dichotic speech but not in complex pitch discrimination, while the right hemisphere-lesioned group was impaired in complex pitch but not speech discrimination. More specifically, patients with unilateral right temporal lobe excisions in the area of Heschl's gyrus and surrounding auditory cortex have been found to be impaired in extracting fundamental pitch from a complex tone (Zatorre, 1988). Further evidence for hemispheric mediation of complex tone discrimination comes from a recent electrophysiological study of normal subjects. Those subjects showing a normal left ear advantage (LEA) on the complex tone discrimination task were found to have greater late event-related potential (ERP) positivity over the right hemisphere than the left hemisphere, while those without a LEA tended to have the opposite ERP asymmetry (Tenke, Bruder, Sidtis, Towey, Erhan & Voglmaier, 1989). These findings suggest that lateralized ear performance can provide valid measures of hemispheric asymmetries for complex pitch perception.

In summary, there is evidence from neurological, neurophysiological and neuropsychological studies which implicate right hemisphere involvement in depression. There is some evidence that right hemisphere dysfunction may be specific to certain subtypes of depression, particularly

patients with melancholic features. There is also some evidence that a dysfunctional left hemisphere may be related to subtype or severity of depression. The few studies of facial emotion processing in depression support abnormalities of expression and appreciation of facial emotion, providing support for hypothesized right hemisphere dysfunction in depression.

2. Anxiety

There are few studies of lateralized emotional processing in the anxiety disorders. In persons who are characteristically anxious, the left hemisphere may be particularly activated (Tucker, 1981). Tucker, Antes, Stenslie, & Barnhardt (1978) found poor right visual field (left hemisphere) performance on both verbal and spatial tasks in trait-anxious college students. These trait-anxious persons were also shown to exhibit a right-ear attentional bias, which was thought to reflect high arousal of the left hemisphere. Additionally, left lateral eye movements were infrequent in trait-anxious subjects. Tucker (1981) has speculated that anxiety may overactivate the left hemisphere to the point of interfering with left hemisphere function, or even producing inhibition of right hemisphere function. In contrast, Davidson and Tomarken (1989) have proposed that individuals with accentuated right anterior EEG activation may be predisposed to withdrawal-related emotions and disorders, such as fear and anxiety disorders, while those

with left anterior hypoactivation may show deficits in approach related emotions, leading to reduced positive affect, sadness, and depression.

More recent evidence has suggested involvement of right temporal-limbic structures in patients with panic disorder. Episodes of panic have been reported in patients with right paralimbic grade-2 astrocytoma (Drubach & Kelly, 1989), right temporal lobe meningioma (Ghardian, Gauthier, & Bertrand, 1986), and right temporal lobe arteriovenous malformation (Wall, Tuchman, & Mielke, 1985). Patients with panic disorder have been found to show more right mesial temporal lobe abnormalities on MRI (Ontiveros, Fontaine, Breton, Elie, Fontaine, & Dery, 1989) and PET scans (Reiman et al., 1984, 1986). A neuropsychological correlate of this has recently been found. Panic disorder patients showed evidence of visuospatial, but not verbal, memory impairment (Lucas, Bigler, & Telch, 1990). Overall, the findings suggestive of temporal lobe dysfunction in panic disorder are consistent with Gray's (1982) theory, which implicates the hippocampus as the neurological substrate of anxiety.

In summary, the few studies available assessing neuropsychological mechanisms underlying anxiety and the anxiety disorders suggest possible left, right, or bilateral hemisphere dysfunction, with more evidence for right temporal lobe involvement in panic disorder per se. There is a clear need for further research into the

neuropsychological mechanisms underlying anxiety-related disorders.

E. Anxious vs. Nonanxious Depression

Evidence for a distinction between anxious and nonanxious depression comes from clinical, epidemiological, and physiological studies which attempt to discriminate anxiety from depressive states for diagnostic purposes. Anxiety and depression constitute the most frequent disorders seen in outpatient psychiatric practice, and their symptoms frequently overlap (Akiskal, 1985), creating difficulties in differential diagnosis, prognosis, and treatment decisions. Laboratory tests like the dexamethasone suppression test (Curtis, Cameron, & Nesse, 1982) and sleep EEG data (Akiskal, Lemmi, Yerevanian, King, & Belluomini, 1982) have differentiated primary depression from primary anxiety states. Watson, Clark, and Carey (1988) found that positive, but not negative, affect distinguished depression from anxiety. They had subjects complete positive and negative trait affect scales, and found that both anxiety and depressive symptoms were associated with high negative affect, but only depressive symptoms were associated with low levels of positive affect. In addition, anxiety states may show greater physiological arousal than depressive states. Lader (1975) found that anxiety and agitated depression increased psychophysiologic activity, while retarded depression decreased it.

Although "anxious-depression" is not a subtyping category recognized by current diagnostic systems, there appears to be a sizable group of classic depressed patients who also report symptoms of worry, anxiety, panic attacks, and phobic and obsessive-compulsive symptoms (Clayton, 1987). A number of phenomenological, biological, and pharmacological differences have been found between anxious and nonanxious depressions, and further distinction of these subtypes is necessary to determine differential etiology, treatment, and prognosis. Patients with anxious depressions have been differentiated from primary dysthymics (subaffective depression) on the basis of sleep parameters (Akiskal, Lemmi, Dickson, King, Yerevanian, & Van Valkenburg, 1984). Anxious-depressed patients have been found to have symptoms that have previously been associated with endogeneity, such as classic diurnal variation (Clayton, 1987). In addition, anxious-depressed patients have been found to have high urinary MHPG excretion, poor response to lithium therapy, later onset of disease, a history of delusions and suicidal ideation (Sachetti, Conte, Calzeroni, Pennati, Battagli, & Vita, 1987), and enlarged cerebral ventricles (Sacchetti, Conte, & Calzeroni, 1987), suggesting that anxious-depression could be seen as an extreme form of a specific subtype of mood disorder, accompanied by cerebromorphological abnormalities. Further evidence for a neurological distinction between anxious and

nonanxious depression comes from a recent study of post-stroke affective disorder, where anxious depression was more often associated with cortical lesions of the left hemisphere, while nonanxious depression was more often associated with subcortical lesions of the left hemisphere (Robinson & Starkstein, 1991).

Patients with both depression and panic attacks have been found to show increased incidence of agitation, hypochondriasis, depersonalization and derealization, chronic depression, and poorer treatment outcome than other depressed patients (e.g., Paykel, Parker, Penrose, & Rassaby, 1979). Van Valkenburg, Akiskal, Puzantian, and Rosenthal (1984) found that that anxious depression, whether defined by the presence of anxiety symptoms or panic attacks, seems to be consistently associated with increased chronicity, poorer response to treatment, and poorer prognosis. Vittone and Uhde (1985) found that patients with both panic and depression tended to have more severe depression with melancholic features, although the depressive episodes tended to be relatively short in duration. Liebowitz et al. (1984) found that atypically depressed patients with a history of panic attacks showed extremely low rates of response to placebo, and high rates of response to an MAOI antidepressant, in comparison to atypically depressed patients without panic attacks.

There is also evidence that depression and panic

disorder specifically are strongly associated and may have common etiological factors. Very high rates of at least one episode of depression have been found among patients with panic anxiety (Bowen & Kohout, 1979; Cloninger, Martin, Clayton, & Guze, 1981; Raskin, Peeke, Dickman, & Pinsker, 1982), and patients with major depression have been found to show higher than chance rates of panic disorder (Leckman, Weissman, Merikangas, Pauls, & Prusoff, 1983). Treatment studies also support the hypothesis of a potential relationship between panic disorder and major depression, in that both disorders are responsive to TCA and MAOI antidepressant medications (Davis, 1980; Breier, Charney, & Heninger, 1985; Sheehan, Ballenger, & Jacobsen, 1980). Family studies have shown increased rates of depression in the relatives of probands with major depression and those with panic disorder, suggesting a link between the two disorders (e.g., Bowen & Kohout, 1979; Leckman et al., 1983). There is some evidence that relatives of patients with both major depression and panic disorder have differential rates of depression than those of patients with major depression alone (Leckman et al., 1983). This data, along with the phenomenological evidence cited above, suggest that individuals with both disorders (i.e., comorbidity of depression and panic disorder) may have a unique syndrome when compared to patients with either disorder alone (Pauls & DiBenedetto, 1987).

F. Summary of the Reviewed Literature

The literature reviewed above provides a rationale for measuring facial EMG responses, the appreciation of facial emotion, and measures of perceptual asymmetry in anxious and nonanxious subtypes of depression. The neuropsychological features of anxious and nonanxious depressive subtypes have not been assessed. However, there is a large body of evidence suggesting that anxious and nonanxious depressions are phenomenologically and biologically distinct, and may have neurological and cerebromorphological differences. Given that the right hemisphere has been implicated in depressive, anxiety and panic disorders, and that the right hemisphere is thought to mediate the expression and appreciation of facial emotion, the measurement of facial emotion processing appears to be a particularly relevant neuropsychological function to assess in these depressive subtypes. In addition, because right temporal and temporo-parieto-occipital areas in particular have been implicated in the perception of faces and facial expressions, and in depressive and panic disorders, measures of auditory perceptual asymmetries appear to be a particularly relevant neuropsychological correlate for the assessment of facial emotion processing.

The current study was designed to be more well-controlled than previous studies assessing facial emotion in depressed individuals. First, the objective methods of

pictures of facial affect and facial EMG were employed to elicit and to measure facial muscle activity, respectively. The use of pictures of facial affect as emotion elicitors also provided a measure of the appreciation of facial emotion, in that subjects rated the valence and intensity of the emotional faces. In addition, psychosocial theories of depression cite deficits in social interactions as a hallmark of depression (e.g, Beck, 1971; Coyne, 1976a, 1976b; Lewinsohn, 1974). Although the present study did not measure real social interaction, because the affective stimuli employed were human faces, it appeared that this paradigm might offer a measure of affective responsivity to social exposure. Second, this study controlled for anxiety in depression by contrasting anxious and nonanxious subtypes, and by employing self-report measures of both anxiety and depression, and assessing their effects on facial muscle activity. Third, the additional neuropsychological measures of verbal and nonverbal dichotic listening measures were employed to provide further support for hemispheric differences that might be involved in anxious and nonanxious depression. Finally, to further delineate the relationship between facial muscle activity and experienced emotion, subjects' immediate subjective feelings and "state" and "trait" anxiety and depression were assessed by self-report measures and related to facial EMG measures. A subgroup of depressed subjects were retested on

the EMG measures following successful response to antidepressant treatment, so as to begin to examine whether EMG alterations in depression are stable characteristics or are related to transient affective states.

The results of Pilot Studies 1 (see Appendix A) and 2 (see Appendix B) supported the feasibility of obtaining reliable measures of facial emotional reactions using the proposed EMG techniques. Preliminary data obtained for depressed patients and normal controls (see Appendix C) were consistent with the hypothesis of group differences in facial EMG activity, and power analyses supported the sample sizes of at least 17 subjects per group.

In the following text, the definition of several key terms is necessary. First, the term "facial expression" is used synonymously with "facial EMG activity". However, it should be noted that the facial EMG activity measured was a covert (e.g., unobservable) physiological response to affective facial stimuli. It is as yet unclear how this activity relates to communicative facial expressions during actual social interactions. Second, the term "immediate emotional experience" is used to mean "reported emotional experience" based on the limited scales (happy or sad) offered to the subjects. Finally, the term "appreciation of facial emotion" is defined as subjects' objective ratings of facial emotion, based on these same limited scales (happy or sad).

G. Statement of Hypotheses

1. Primary Hypotheses

a. Anxious depression involves greater dysfunction of the right hemisphere than does nonanxious depression.

If this hypothesis is correct:

i. Anxious depressed subjects will show greater abnormalities in the expression of facial emotion (as measured by facial EMG patterns) in comparison to nonanxious depressed subjects.

ii. Anxious depressed subjects will show greater abnormalities in the appreciation of emotion (as measured by objective ratings of valence and intensity of affective faces) in comparison to nonanxious depressed subjects.

iii. Anxious depressed subjects will show greater evidence of right hemisphere dysfunction on dichotic listening tasks (specifically, a larger right ear advantage on the verbal task and a smaller left ear advantage on the nonverbal task) in comparison to nonanxious depressed subjects.

b. Depression involves right hemisphere dysfunction.

If this hypothesis is correct:

i. Depressed subjects in general will show abnormalities in the expression of facial emotion (as measured by facial EMG patterns) in comparison to normal control subjects.

ii. Depressed subjects will show abnormalities in the

appreciation of emotion (as measured by objective ratings of valence and intensity of affective faces) in comparison to normal control subjects.

iii. Depressed subjects will show evidence of right hemisphere dysfunction on dichotic listening tasks (specifically, a larger right ear advantage on the verbal task and a smaller left ear advantage on the nonverbal task) in comparison to normal control subjects.

2. Secondary Hypotheses

a. The corrugator muscle is the effector of "negative" affect, and the zygomatic muscle is the effector of "positive" affect.

If this hypothesis is correct,

i. Normal control subjects will show greater corrugator activity and greater experience of sadness in response to sad faces, and greater zygomatic activity and greater experience of happiness in response to happy faces.

ii. Depressed subjects will show greater corrugator activity and lower zygomatic activity than nondepressed subjects.

iii. The resolution of depression will be accompanied by a reduction in corrugator muscle activity, an increase in zygomatic muscle activity, and a normalization of abnormal muscle activity patterns.

iv. Measures of immediate subjective experience of sadness, and measures of state and trait depression and

anxiety will be associated with increased corrugator activity.

v. Measures of immediate subjective experience of happiness will be associated with increased zygomatic activity.

vi. Measures of social, but not physical, anhedonia will be associated with reduced zygomatic muscle activity in response to happy faces.

b. Expressed and experienced emotion are "intimately" related.

If this hypothesis is correct, then because of the temporal proximity between facial EMG activity and subjective emotional experience,

i. Measures of immediate affective experience will correspond to patterns of facial muscle activity.

ii. Measures of immediate affective experience will show stronger associations to facial muscle activity than "state" and "trait" measures of affect, and measures of "state" affect will show stronger associations to measures of facial muscle activity than measures of "trait" affect.

Method

A. Subjects

Because Schwartz et al. (1980) found that females had higher EMG magnitude than males, in this experiment, only females were studied to avoid possible sex differences.

1. Patients

The patient sample consisted of 34 female depressed patients, mainly with atypical features (30 patients were diagnosed as having an atypical depression [Liebowitz, et al., 1988] 3 patients had only the core symptom of mood reactivity, and 1 patient had melancholic features), who were attending an outpatient depression research clinic at the New York State Psychiatric Institute. All aspects of diagnostic assessment and drug treatment of patients were carried out by research psychiatrists on this unit. All patients met DSM-IIIr criteria for a depressive disorder (30 with a major depressive disorder [MDD], 4 with dysthymic disorder [DD]). They were divided into two subgroups: a) the Anxious Depression group consisted of 17 patients with both a depressive disorder (15 MDD, 2 DD) and a DSM-IIIr anxiety disorder (panic disorder, social phobia, or generalized anxiety disorder) or panic attacks which did not meet the frequency criteria for DSM-IIIr panic disorder; and b) the Nonanxious Depression group consisted of 17 patients with a depressive disorder (15 MDD, 2 DD) but no anxiety disorder. All patients in the anxious depression group had some form

of panic anxiety (13 with panic disorder, 4 with panic attacks) and 7 patients had additional diagnoses of anxiety disorders (4 with social phobia, 2 with generalized anxiety disorder, 3 with agoraphobia).

2. Normal Controls

A normal control group of 20 paid volunteers was recruited from New York State Psychiatric Institute staff, areas surrounding the hospital, and from local colleges. They were interviewed using a modified version of the Schedule for Affective Disorders and Schizophrenia--Lifetime Version (SADS-L; Spitzer & Endicott, 1978). No normal control subjects had a current mood or anxiety disorder. Four subjects had a past history of one episode of major depression. None of these subjects had a major depressive episode lasting longer than two weeks, and none had had a recurrent episode. No normal control subjects had a past history of anxiety disorder or other psychiatric disorder (e.g., schizophrenia, personality disorder). Subjects with a history of neurological or neuromuscular disorder were excluded from participation in the study.

Table 1 shows the demographic characteristics of the subject groups. Because of a high rate of left-handedness among atypically depressed patients (Bruder et al., 1989) both right- and left-handed subjects were included in the study. Approximately equal proportions of non-right-handed subjects were included in each group. The Normal Control

Table 1. Subject Characteristics.

		<u>Normal Controls</u>	<u>Anxious Depression</u>	<u>Nonanxious Depression</u>	<u>p</u>
N		20	17	17	
Non R-handed		4	2	3	NS*
Handedness $\bar{X}$		55.25	65.20	53.34	NS
LQ	(<u>SD</u>)	(59.94)	(60.35)	(60.08)	
Age	$\bar{X}$	32.00	39.71	34.94	.06
	(<u>SD</u>)	(10.04)	(8.45)	(10.46)	
Education	$\bar{X}$	17.00	16.47	15.82	NS
	(<u>SD</u>)	(3.20)	(2.58)	(1.78)	
BDI	$\bar{X}$	1.90	25.29	18.25	< .00001
	(<u>SD</u>)	(1.92)	(11.73)	(10.66)	NC< AD = ND

* based on chi-square; all others based on F, one-way ANOVA

group included 16 right-handed subjects, three left-handed subjects, and one ambidextrous subject; the Nonanxious Depression group included 14 right-handed subjects, two left-handed subjects, and one ambidextrous subject; the Anxious Depression group included 15 right-handed and two left-handed subjects. Handedness was assessed by laterality quotient (LQ) scores on the Edinburgh Handedness Inventory (Oldfield, 1971; see Appendix D). A separate question sheet was attached to ensure that subjects did not switch handedness at an early age. The groups did not differ significantly in mean handedness LQ, age, or education, although there was a trend for anxious depressed patients to be slightly older than the other groups (about 7 years on average). The Beck Depression Inventory (BDI; Beck, 1978) was employed as a measure of severity of depression. The patient groups were significantly more depressed than the control group, $F(2,52) = 33.82$, $p < .00001$. The two patient groups did not differ in severity of depression as measured by the BDI.

All participants were given an audiometric screening test. Two normal controls and one patient showed a hearing loss (greater than 30 dB SPL above normative values in either ear at 500, 1000, or 2000 Hz), and were not administered the dichotic listening tasks. Three additional patients were not administered the dichotic tasks because of time constraints. Subjects were screened for normal or

corrected-to-normal vision. They were naive as to the exact purpose of the study. All subjects gave informed consent (see Appendix E) and were paid for their participation.

B. Facial EMG Task

1. Apparatus & Stimuli

Facial EMG Recording. Because of the uncertainties regarding lateralized EMG measurement of facial asymmetries (Fridlund, 1988), only left-sided facial regions were studied to control for laterality effects. Bipolar EMG was recorded from the left corrugator and zygomatic muscle regions of the face, using Beckman miniature (.25 cm) Ag/AgCl surface electrodes. Electrodes were placed over the two muscle regions according to the standard placements found by Tassinary, Cacioppo, & Geen (1989) to be the most sensitive, valid and reliable for differential EMG recording. Over the corrugator muscle region, the first electrode was placed directly above the eyebrow, vertical to the endocanthion, and the second was placed 1 cm lateral and slightly superior to the first. Over the zygomatic muscle region, the two electrodes were placed between 2.5 and 4.5 cm from the corner of the mouth (cheilion), along an imaginary line connecting the cheilion and the preauricular depression. A single ground electrode was placed on the center of the forehead. To divert attention away from the face, "dummy" electrodes were placed on the left and right hand, because such demand characteristics have been found to

influence EMG activity levels (Fridlund & Cacioppo, 1986).

Facial EMG was recorded via a Grass Model 12 Neurodata Acquisition system. The EMG signal was amplified by Grass amplifiers, with high- and low-pass filters set at 30 and 300 Hz, respectively. A line filter was employed to exclude 60-cycle noise. The signal was amplified 50,000 times, and was recorded at a rate of 600 samples per second. The signal amplitude was digitized, and the root mean square (RMS) of the waveform was calculated in one second epochs. Because the RMS has been found to provide the most accurate index of underlying physiological events (Basmajian & DeLuca, 1985), it was employed for processing and quantifying the EMG amplitude. This measure computes the square root of the mean of squared EMG samples, and is therefore equivalent to a measure of standard deviation.

Affective Stimuli. The faces used as stimuli were 36 black and white slides selected from Ekman's "Pictures of Facial Affect" (1975) showing male and female adults producing 12 neutral, 12 happy, and 12 sad facial expressions. These affective stimuli were used in order to parallel previous research employing facial EMG in depressed subjects (e.g., the happy, sad, and "typical day" imagery used by Schwartz, et al., 1976a, and Greden, et al., 1986). Additionally, a previous pilot study using control subjects had shown the expected EMG patterns in response to these stimuli (see Appendix A). The slides were selected by the

experimenter for high affective intensity. Slides were grouped into "affective" blocks (neutral, happy, sad), with a random order of 6 male and 6 female posers. Each block of 12 affective slides was preceded by a set of 6 "blank" slides, used as a baseline for that block. Blanks were a translucent gray color, with approximately the same luminance as the affective slides. During the 16 s interstimulus interval, subjects were exposed to a gray translucent background.

Objective Ratings and Subjective Experience of Emotion.

The use of a 5-point Likert scale (see Appendix F) was found to yield scores of sufficient variability in Pilot Study 1 (see Appendix A), and was again employed in the present study. Subjects were given a single sheet of paper showing two printed 5-point Likert scales, labeled "Happy" and "Sad", respectively. They were asked to rate each slide for affective intensity ("objective" ratings, a measure of appreciation of facial emotion) and to rate the affective intensity of their own feelings during exposure to each slide ("subjective" ratings, a measure of immediate emotional experience). Ratings consisted of choosing either "Happy" or "Sad", and choosing a number from 1 (low intensity) to 5 (high intensity).

Self-Report Measures of Emotion. In order to assess the relationship between expressed (as measured by facial EMG amplitude) and experienced emotion, several self-report

measures of emotion experience were employed. These measures included the a) the Depression scale scores from the Multiple Affect Adjective Check List-Revised (MAACL-R; Zuckerman & Lubin, 1985; see Appendix G), which yielded a measure of trait depression; b) the Depression Adjective Checklist (DACL; Zuckerman & Lubin, 1981; see Appendix H) which provided a measure of state level of depressed affect; and c) the State-Trait Anxiety Inventory (STAI; Spielberger, 1983) a widely used inventory which provided measures of both state and trait anxiety levels (see Appendix I); d) the Revised Social Anhedonia Scale (Chapman, Chapman, & Raulin, 1976; see Appendix J); and e) the Revised Physical Anhedonia Scale (Chapman et al., 1976; see Appendix K). The last two measures were included based on the findings of Pilot Study 1 (see Appendix A), in which high trait depression was found to be associated with the attenuation of both zygomatic muscle activity and subjective experience of happiness in response to happy faces. It was thought that this might represent a physiological correlate of "anhedonia", or reduced capacity to obtain pleasure from "pleasurable" stimuli.

2. Procedures

Subjects gave informed consent, and completed the self-report measures of emotion prior to electrode placement. All aspects of electrode placement took place in a room separate from the experimental chamber. In the experimental chamber,

subjects were seated in an adjustable dentist chair (Dynadust), about 80 cm from a Raven plexiglass rear projection screen (Model LS 60). A curtain was drawn around the screen to provide a uniform background. Subjects were given the sheet containing the Likert scales, and were instructed (see below). In order to habituate to the chamber, subjects rested for 5 minutes prior to beginning the session. Five minute rest periods were given between blocks of slides. Prior to slide onset, subjects heard a warning tone via a mini amplifier/speaker (Archer), to alert them to watch the screen. Slides were projected via a Kodak Ektagraphic IIIIB projector. Slide onset and duration was controlled by a Dell System 200 computer. About 1 s after the tone, a 15.5 cm X 24 cm slide image appeared on the screen for 4 s. The interstimulus interval was 16 s. The experimenter sat in an adjacent room, and monitored subjects on a television monitor (Ampex), via a closed circuit television camera (General Electric, Model 500). Trials with overt facial expressions were noted, and were excluded from the analysis at a later time. All communication between the subject and experimenter took place via a Fanon (Model IN-400) selective 2-way monitor situated next to the subject. Following each slide presentation, subjects vocally rated the face for affective intensity and immediate subjective experience of happiness or sadness, according to the two Likert scales. The order of the affective blocks (neutral,

happy, sad) was randomly alternated among subjects.

Subject Instructions. Subjects were instructed as follows: "We will be measuring physiological activity while you look at slides on the screen. Some of the slides are blank, and some show pictures of faces. Before each slide, you will hear a warning tone. This is your cue to watch the screen. Each slide will stay on the screen for about 5 seconds, and you are to look at each slide for the full 5 seconds. You can rest between slides. Over the intercom, I will ask you to rate each face for its emotion, and the intensity of that emotion based on these two scales. You must choose either "Happy" or "Sad", then give a number for how intensely the emotion was expressed on the face, where 1 is very weak, and 5 is very strong. Then I'll ask you how you felt while the picture was on the screen. Again, you must choose either happy or sad, and give a number from 1 to 5, depending on how intense your feelings are at that time. Try to be as accurate as you can about how you feel at that time."

C. Dichotic Listening Tasks

1. Apparatus & Stimuli

Consonant-Vowel (CV) Task. A tape recording of the CV discrimination task was provided by the Kresge Hearing Research Laboratory. The CV task consisted of three lists of 30 randomly paired stop-consonant-vowels (ba, da, ga, ta, pa, ka). Each subject received 16 monaural trials (to check

accuracy of CV identification for each ear), 14 dichotic practice trials, and 60 dichotic test trials. Dichotic trials consisted of presenting a different syllable simultaneously to each ear. The subject reported the two syllables presented using a multiple choice answer sheet. The answer sheet contains a random ordering of the six possible syllables for each trial. The subject was instructed to cross off the two syllables that were heard on each trial and to guess where necessary. This task has consistently yielded a mean right-ear (left hemisphere) advantage for groups of normal right-handed children and adults (Berlin, Hughes, Lowe-Bell, & Berlin, 1973; Berger-Gross and Bruder, 1984; Speaks, Niccum, & Carney, 1982). Other procedural details are described in Berlin et al. (1973). The tape was presented to subjects by a stereo reel to reel tape deck (TEAC) and matched ear phones (TDH-49). A 1,000-Hz reference tone at the beginning of the tape was used in calibrating the output of each earphone with a .5 in (1.27 cm) Bruel and Kjaer microphone. The tape was presented at a comfortable level of 75 dB (SPL).

Complex Tone (CT) Test. This test required subjects to compare the pitch of a binaural complex tone to the pitches of a dichotic pair of complex tones presented 1 s earlier. The complex tones were square waves with fundamental frequencies corresponding to the eight notes in the octave between C4 and C5 on the major scale. Subjects responded

"yes" when the probe tone was the same as either member of the previous dichotic pair, or "no" when it differed from both. After 16 practice trials, each subject was tested on four blocks of 28 trials each (in some cases, subjects were tested on 6 blocks of 28 trials, because they were also involved in another study). The complex tone test was presented at 75 dB. This task has consistently yielded a mean left ear (right hemisphere) advantage in normal adults (Bruder, et al., 1989; Sidtis, 1981).

2. Procedures

Subjects sat in a soundproof chamber during administration of the dichotic listening tasks. In most cases, depending on availability of equipment, the order of administration of the two tasks was alternated among subjects to control for order effects. The position of the earphones was also alternated among subjects, to control for possible differences in quality of sound.

D. Retest Procedures

A subgroup of 17 patients (8 anxious depressives and 9 nonanxious depressives) who responded to antidepressant treatment (as assessed by research psychiatrists) and all normal control subjects were retested on the facial EMG tasks, with the same order of affective blocks as in the initial session. An alternate version of the DACL and the "state" measure of the STAI was administered to provide measures of current ("state") depression and anxiety.

Retests of patients and controls took place after similar time intervals (approximately 6 to 12 weeks).

E. Data Reduction and Analysis

1. Facial EMG Task

EMG data were log-transformed prior to averaging to improve the normality of the data and to correct for differences in variances across groups (Gaddum, 1945). Data collected during exposure to "blank" slides was averaged, and the mean of each set of "blanks" was used as a baseline measure for each set of affective slides. There were no differences between the groups in baseline EMG patterns for either corrugator, $F(4,102) = 1.85$, NS, or zygomatic, $F(4,102) = .79$, NS, muscle activity. Data collected during exposure to sets of affective slides was averaged, to yield a mean EMG magnitude for each muscle (corrugator, zygomatic) during exposure to each of the three affective conditions (neutral, happy, sad). Analyses of covariance (ANCOVAs), using the baseline measures as covariates, were employed, so that variations in baseline EMG levels among the groups or across conditions did not skew observed patterns of muscle response. In order to determine facial EMG pattern differences between the experimental groups, analyses included a 3 X 2 X 3 repeated measures factorial ANCOVA, with one "between groups" factor, Group (Anxious, Nonanxious, Normal Control), and two "within groups" factors: Muscle (corrugator, zygomatic) and Affective

Condition (neutral, happy, sad). To further determine individual group differences, Group X Affective Condition ANCOVAs were conducted for each muscle, and simple effects analyses were conducted to determine where specific differences in EMG magnitude lay. Post-hoc analyses of individual means employed the Tukey HSD procedure, with alpha set at .05. To determine differences in facial EMG for depressed subjects in general and normal controls, similar analyses were performed for these two groups. The dependent variable in each of these analyses was the log-10 transformed RMS EMG values, measured in microvolts.

The results of Pilot Study 2 (see Appendix B) did not reveal the expected corrugator magnitude differences for sad and neutral faces. Because emotion-dependent differences in the "growth" of corrugator and zygomatic muscle activity had been found in Pilot Study 1 (Appendix A), and were reported in the literature (e.g., Sirota, Schwartz, & Kirsteller, 1987), time course effects were also assessed. This was accomplished in two ways: a) by taking averages of each second of the 4 second exposure period during each affective condition, and b) by taking averages of the first and second half (e.g., 6 slides each) of each affective condition. This procedure provided measures of both "within-slide" and "within-block" time course changes. Time course effects were compared between groups for each muscle during each affective condition by ANCOVAs, in order to provide more

specific information on differences in facial muscle activity patterns between the groups.

To determine group differences in the appreciation of facial emotion, measures of objective ratings of valence and intensity of the affective faces were subjected to Group X Affective Condition repeated measures analyses of variance (ANOVAs).

To evaluate group differences in self-reported immediate subjective experience of emotion in response to pictures of facial affect, subjective ratings of valence and intensity of affect were subjected to Group X Affective Condition ANOVAs. In order to determine the association between expressed (facial EMG activity) and experienced emotion, two-tailed Pearson product-moment correlations were performed between these self-report measures and facial EMG measures for each affective condition.

In order to assess the association between levels of depression, anxiety, anhedonia and patterns of facial EMG magnitude, the self-report measures were correlated with EMG measures in each affective condition. To assess changes in patterns of facial muscle response as a result of antidepressant treatment response, the initial and retest EMG values of those patients who responded to treatment and normal controls were subjected to a Group X Session X Muscle X Affective Condition ANCOVA.

2. Dichotic Listening Tasks

Data from the CV and CT tasks were analyzed using absolute accuracy scores for each ear, as well as a measure of perceptual asymmetry. Right (R%) and left (L%) ear accuracy scores for the CV task were obtained by computing the percentage of correct responses for stimuli presented to each ear. Accuracy scores for the CT task were obtained by computing the correct percentage of "YES" responses separately for stimuli presented to each ear. The laterality index $\underline{e}$ (Halwes, 1968; Repp, 1977) was used as the measure of perceptual asymmetry as it controls for laterality differences being a reflection of performance level (Bryden, 1982, p. 32). The $\underline{e}$ index is calculated separately for overall performance levels greater than 50%, $\underline{e} = (R\% - L\%)/(200 - R\% - L\%)$, and less than 50%, $\underline{e} = (R\% - L\%)/(R\% + L\%)$. This index has limits of -1 and +1, with a positive number indicating a right ear advantage, and a negative number indicating a left ear advantage.

Laterality index scores for the two tasks were subjected to a Group X Task ANOVA to determine group differences in perceptual asymmetry for the nonverbal and verbal dichotic tasks. A Group X Task X Ear ANOVA was conducted to assess accuracy differences in ear performance between the groups. Both the laterality index and accuracy scores from the dichotic tasks were correlated with EMG measures for each group, to assess the relationship between perceptual asymmetry and facial muscle activity patterns.

Results

A. Comparison of Anxious vs. Nonanxious Depression vs. Normal Control Groups

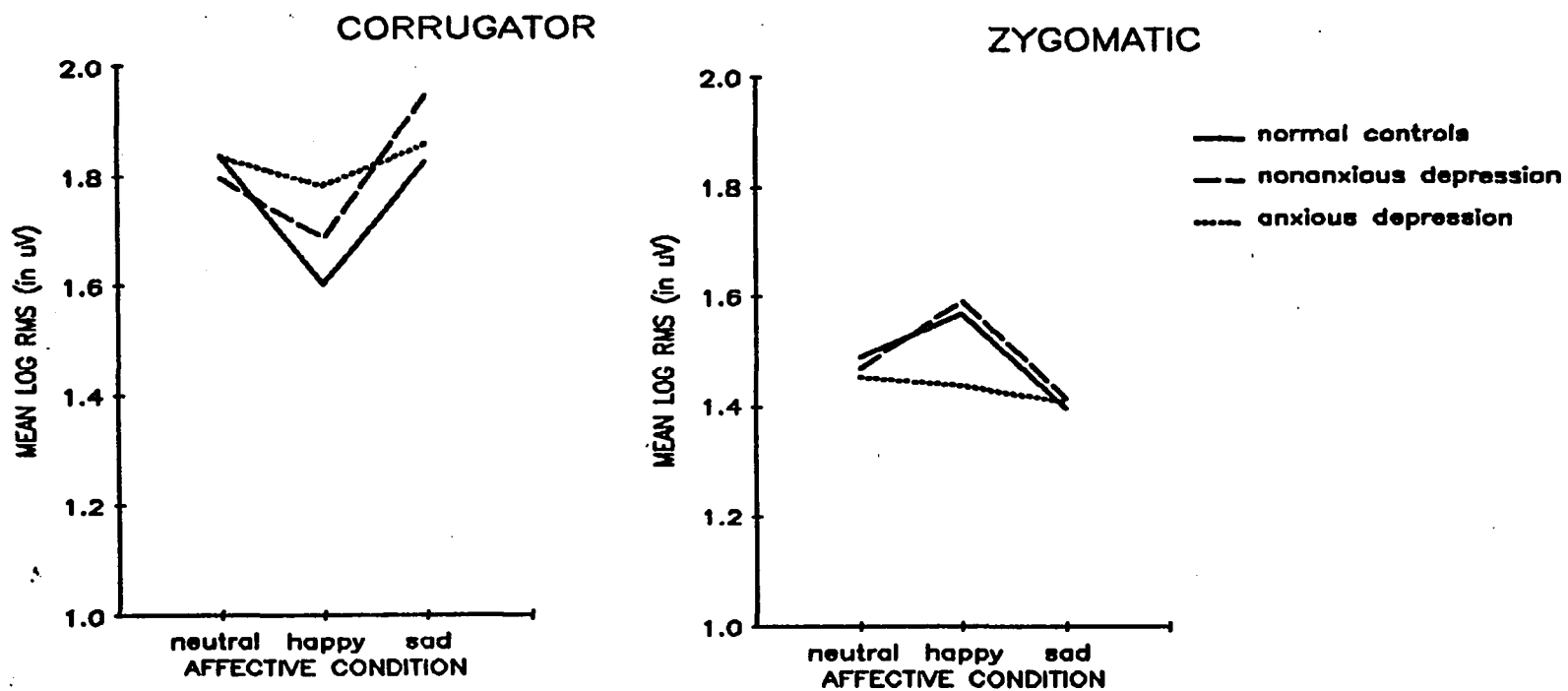
To test the first hypothesis, that anxious depression is characterized by more right hemisphere dysfunction than nonanxious depression, the Anxious Depression, Nonanxious Depression, and Normal Control groups were compared on measures of expression (i.e., facial EMG activity) and appreciation (i.e., objective ratings) of facial emotion and the dichotic listening tasks.

1. Expression of Facial Emotion

a. Facial EMG Magnitude. In order to examine facial muscle patterns in the experimental groups, facial EMG measures were subjected to a Group X Muscle X Affective Condition ANCOVA. Results of this analysis showed a highly significant Muscle X Affective Condition interaction, $F(2,101) = 37.75$, $p < .00001$, indicating that the corrugator and zygomatic muscles exhibited different patterns of facial EMG magnitude in response to affective faces (Figure 2). The 3-way interaction was also significant, $F(4,101) = 3.23$, $p < .02$, indicating that these patterns differed for the experimental groups. There were no other significant main effects or interactions.

Separate Group X Affective Condition ANCOVAs were employed for the corrugator and zygomatic muscles, to further determine the EMG pattern specific to each muscle.

Figure 2. Mean corrugator and zygomatic muscle EMG magnitude for Anxious and Nonanxious Depressed subjects and Normal Controls.



Corrugator. For the corrugator muscle, the Group X Affective Condition interaction indicated a significant difference in corrugator muscle patterns among the subject groups, $F(2,103) = 2.51, p < .05$. One-way ANCOVAs conducted for each subject group revealed that both the Normal Control [$F(2,37) = 11.06, p < .001$] and the Nonanxious Depression [$F(2,31) = 16.93, p < .00001$] groups showed highly significant differences in corrugator response to the different affective faces. In contrast, the Anxious Depression group showed no difference in corrugator activity as a function of affective condition, $F(2,31) = 2.19, NS$. Post-hoc Tukey HSD analyses of the individual means revealed that both the Normal Controls and the Nonanxious Depressed patients showed reduced corrugator activity for happy faces in comparison to neutral and sad faces ($p < .05$), the Normal Controls showed equal corrugator magnitudes for neutral and sad faces, but the Nonanxious Depressed patients showed greater corrugator magnitude for sad faces than for neutral faces ($p < .01$). Additionally, the Anxious Depression group showed significantly greater corrugator EMG ($\underline{M} = 1.78, \underline{SD} = .35$) magnitude during exposure to happy faces than did Normal Controls ($\underline{M} = 1.60; \underline{SD} = .25$), $p < .05$. The Nonanxious Depression group did not differ from either of the other subject groups in corrugator response to happy faces. There were no differences between the groups in corrugator response to neutral or sad faces.

Zygomatic. Analysis of zygomatic muscle responses also revealed a significant Group X Affective Condition interaction, $F(2,101) = 2.67$, $p < .05$. Post-hoc analyses showed that both the Normal Control and Nonanxious Depression groups exhibited greater zygomatic activity in response to pictures of happy faces than to pictures of sad and neutral faces ($p < .01$). The zygomatic responses of these groups to pictures of sad and neutral faces were not significantly different. In contrast, the Anxious Depression group showed no significant differences in zygomatic response to the affective faces, $F(2,31) = 1.12$, NS. Additionally, the Anxious Depression group showed significantly lower zygomatic magnitude ($\underline{M} = 1.43$, $\underline{SD} = .24$) in response to happy faces when compared to the Nonanxious Depression group ($\underline{M} = 1.59$, $\underline{SD} = .28$), ($p < .05$). The zygomatic response of the NC group ($\underline{M} = 1.57$, $\underline{SD} = .24$) to pictures of happy faces did not differ from either of the two patient groups. The groups did not differ in zygomatic response to neutral or sad faces.

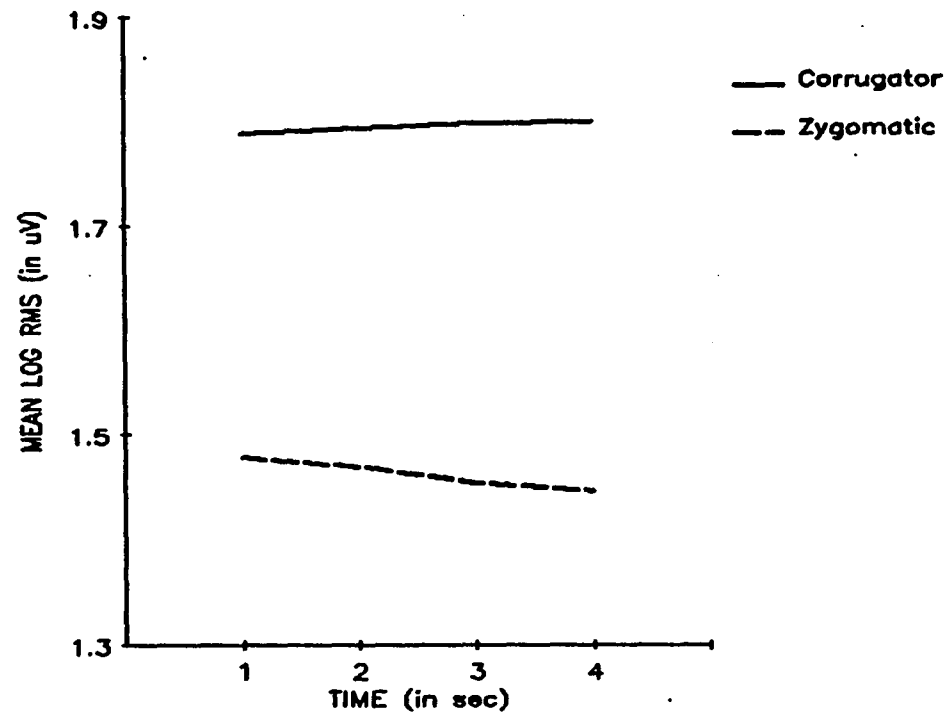
Controlling for Age Effects. Because of a trend for age differences between the experimental groups, Pearson product-moment correlations were conducted between age and EMG activity. No systematic relationship was found between these variables. Significant correlations were found between age and zygomatic activity during the neutral ($r = .54$, $p < .05$) and sad ($r = .53$, $p < .05$) condition for the Nonanxious

Depression group. No significant correlations were found between age and EMG activity for either the Anxious Depression or Normal Control groups. Results of Group X Affective Condition ANCOVAs, with age as the covariate, revealed the same significant Group X Affective Condition interaction for corrugator, $F(4,101) = 2.51, p < .05$, and zygomatic, $F(4,101) = 2.67, p < .05$, muscle activity as in the original analyses (see Appendix L).

b. Time Course of Facial EMG. Significant differences were not found between the magnitude of corrugator activity for neutral and sad faces for normal controls. Based on the results of Pilot Study 1 (see Appendix A), and two studies which found emotion-dependent time course differences in corrugator and zygomatic muscle activity (Sirota & Schwartz, 1982; Sirota, Schwartz, & Kirsteller, 1987), the time course of facial EMG patterns was assessed. It was predicted that corrugator activity would show significant increases over time, particularly during exposure to sad faces.

Two different approaches were used to examine changes in time course of facial EMG responses. The first assessed muscle responses during the 4 s of slide presentation ("within slide" time course). The results of the overall Group X Muscle X Affective Condition X Time ANCOVA revealed a highly significant Muscle X Time interaction, $F(3,152) = 24.99, p < .00001$. The "within slide" time course for the two muscles is shown in Figure 3. Post-hoc Tukey tests

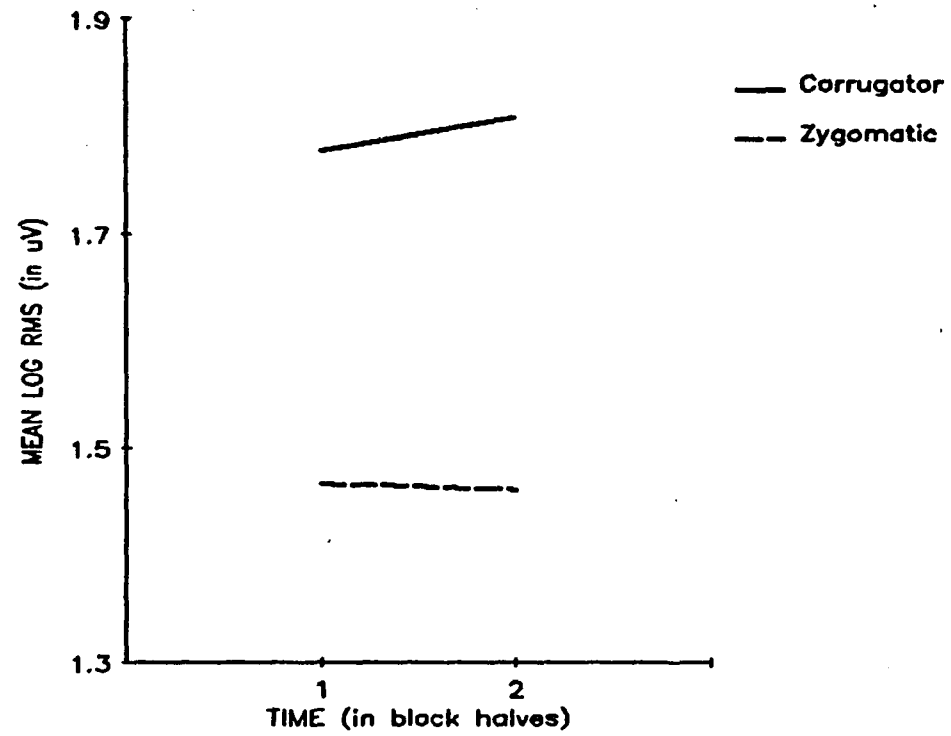
Figure 3. Mean muscle time course changes across the 4 second stimulus presentation time period ("within-slide").



revealed that while the corrugator showed a significant increase from the first to the fourth second of slide presentation ($p < .05$), the zygomatic muscle decreased significantly from the first to the third ($p < .05$) and fourth ($p < .01$) second. There were no other significant interactions with Time, indicating no differences in "within slide" time course between groups, or among affective conditions.

The second approach to assessing time course changes examined EMG differences from the first to the second half (six slides per half) of each affective block of slides ("within block" time course). The results of the overall Group X Muscle X Affective Condition X Time ANCOVA again revealed a significant Muscle X Time interaction, $F(1,51) = 6.55$, $p = .01$, indicating "within block" time course differences for the corrugator and zygomatic muscles (see Figure 4). Individual analyses for each muscle revealed that while the corrugator showed a significant increase in magnitude from the first ($\bar{M} = 1.78$) to the second ($\bar{M} = 1.81$) half of each affective block [$F(1,51) = 7.32$, $p < .01$], the zygomatic muscle did not differ in magnitude from the first to the second half of each block [$\bar{M} = 1.47$ vs. $\bar{M} = 1.46$; $F(1,51) = .80$, NS]. There were no other significant interactions with Time, indicating no differences in "within block" time course between the subject groups, or among affective conditions.

Figure 4. Mean muscle time course changes across stimulus block halves ("within-block").



2. Appreciation of Facial Emotion

In order to determine whether group differences existed in appreciation of facial affect, subjects' objective emotion ratings of the slides were subjected to a Group X Affective Condition ANOVA. Intensity ratings on the "Happy" scale were scored as positive numbers (i.e., +1 to +5 for judgments of low to high intensity of happy affect, respectively), and those on the "Sad" scale were scored as negative numbers (i.e., -1 to -5 for low to high intensity of sad affect, respectively). The results of this analysis can be seen in Figure 5; the individual means are listed in Table 2. The main effect of Affective Condition was highly significant, $F(2,102) = 344.61$, $p < .00001$. Post-hoc Tukey HSD tests revealed that overall, subjects rated happy faces as more happy than neutral and sad faces, sad faces as more sad than neutral and happy faces, and neutral faces as less happy than happy faces and less sad than sad faces ($p < .01$). The interaction was not significant, indicating no differences between the subject groups in their ability to judge the valence and intensity of facial affect.

3. Auditory Perceptual Asymmetry

The asymmetry ($\bar{e}$) and accuracy scores for the verbal and nonverbal dichotic listening tasks were analyzed separately for all subjects (i.e., right- and left-handed), and for right-handed subjects only. Individual means for the full groups and for right-handed subjects only are presented

Figure 5. Mean objective emotion ratings (facial appreciation).

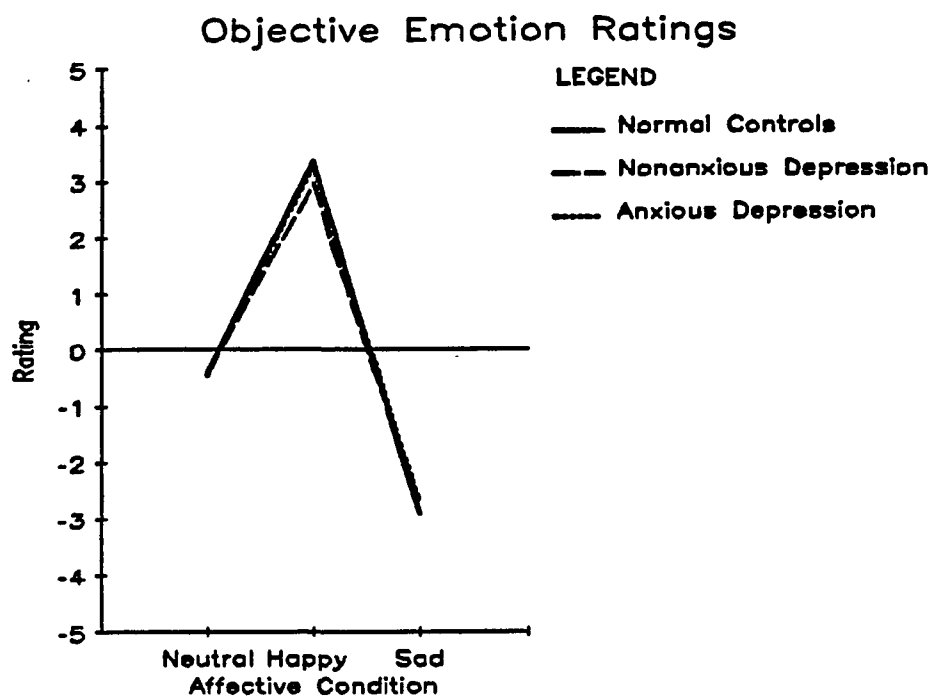


Table 2. Mean Objective Facial Emotion Ratings.

		<u>Normal Controls</u>	<u>Anxious Depression</u>	<u>Nonanxious Depression</u>	p
Affective Condition					
Neutral	<u>X</u>	-.42	-.45	-.44	NS
	(SD)	(1.15)	(1.05)	(.91)	
Happy	<u>X</u>	3.36	3.25	2.94	NS
	(SD)	(.70)	(.94)	(1.50)	
Sad	<u>X</u>	-2.90	-2.68	-2.75	NS
	(SD)	(.68)	(1.15)	(1.75)	

in Table 3. Although the laterality effects were in the same direction for both analyses, handedness did affect the strength of these effects. For this reason, only the results of the analyses for right-handed subjects will be presented here. [Because they obtained perfect scores on the nonverbal complex tone task, asymmetry scores could not be calculated for one Normal Control and one Anxious Depressed subject; however, these subjects were included in the analyses of accuracy scores].

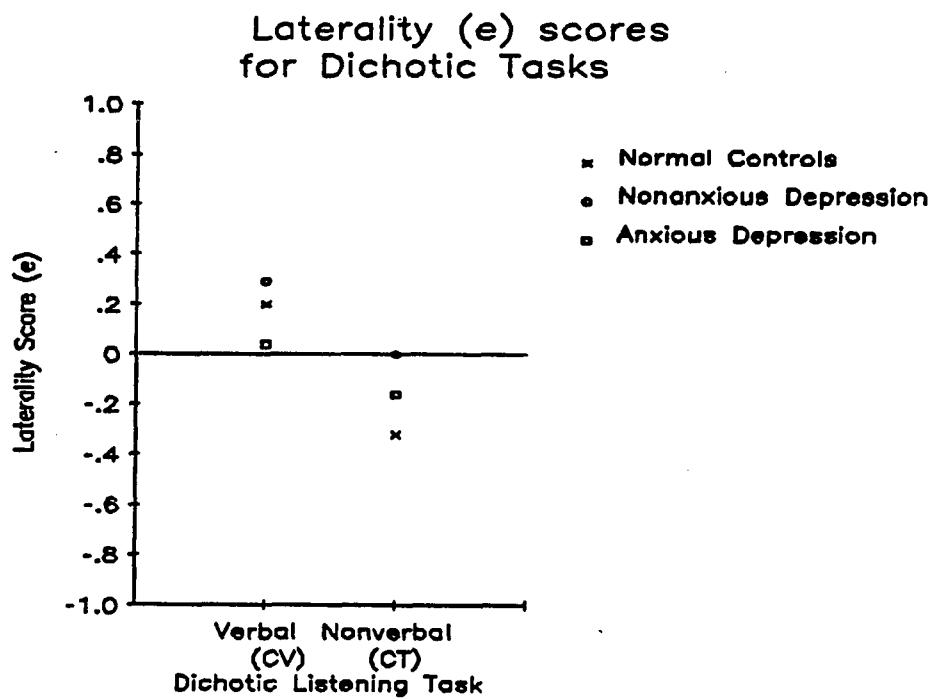
a. Asymmetry (e) Scores. Asymmetry (e) scores on the verbal and nonverbal dichotic listening tasks were subjected to a Group X Task ANOVA (see Figure 6). Results of this analysis revealed a highly significant Task effect $F(1,35) = 13.36$, $p < .001$, with an overall right ear advantage (REA) on the verbal task ($\bar{M} = 0.18$), and an overall left ear advantage (LEA) on the nonverbal task ($\bar{M} = -.16$). The main effect of Group, and the Group X Task interaction, were not significant. However, visual inspection of Figure 6 revealed some differences in the group means, which warranted further analyses.

Individual one-way ANOVAs were performed for each task. Analysis of the verbal CV task indicated a significant Group difference, $F(2,39) = 3.76$, $p = .03$. Post-hoc Tukey tests revealed that the REA of the Anxious Depression group ($\bar{M} = .04$) was significantly smaller than that of the Nonanxious Depression group ($\bar{M} = .29$) [$p < .05$]. The mean asymmetry

**Table 3. Dichotic Listening Performance for Full Group
and Right-Handed Subjects Only.**

		<u>Normal Controls</u>		<u>Anxious Depression</u>		<u>Nonanxious Depression</u>	
		<u>RH+LH</u>	<u>RH</u>	<u>RH+LH</u>	<u>RH</u>	<u>RH+LH</u>	<u>RH</u>
N		18	14	15	14	15	12
<u>Verbal (CV) Task</u>							
Right ear %	<u>X</u>	68.93	72.52	65.57	66.80	74.82	75.84
	(<u>SD</u>)	(17.22)	(14.84)	(14.57)	(14.31)	(15.22)	(14.67)
Left ear %	<u>X</u>	63.27	60.84	62.23	63.94	59.56	58.59
	(<u>SD</u>)	(14.82)	(14.67)	(14.76)	(13.70)	(12.38)	(13.27)
R% - L%	<u>X</u>	5.66	11.68	3.34	2.86	15.26	17.26
e	<u>X</u>	.11	.20	.05	.04	.27	.29
	(<u>SD</u>)	(.30)	(.19)	(.26)	(.27)	(.27)	(.27)
<u>Nonverbal (CT) Task</u>							
Right ear %	<u>X</u>	75.92	76.70	79.21	79.76	83.89	84.32
	(<u>SD</u>)	(21.74)	(21.11)	(14.66)	(15.04)	(11.28)	(10.81)
Left ear %	<u>X</u>	86.90	86.22	83.97	83.50	86.90	86.61
	(<u>SD</u>)	(13.22)	(14.52)	(12.58)	(12.92)	(9.89)	(9.26)
R% - L%	<u>X</u>	-10.98	- 9.52	-4.76	-3.74	-3.01	-2.29
e	<u>X</u>	-.32	-.32	-.18	-.16	.02	.00
	(<u>SD</u>)	(.46)	(.52)	(.53)	(.54)	(.59)	(.55)

Figure 6. Mean asymmetry (e) scores for dichotic listening tasks.



scores of the depressed groups were not significantly different from that of the Normal Control group ($M = .20$). However, results of a Pearson chi-square analysis revealed that in comparison to the Normal Control and Nonanxious Depression Groups, significantly fewer subjects in the Anxious Depression group showed a REA on this verbal task, $X^2(2) = 8.18$, $p = .02$. Only 36% (5/14) of subjects in the Anxious Depression group showed an REA the CV task (8 subjects showed an LEA, one showed no ear advantage), while 83% (10/12) of subjects in the Nonanxious Depression group, and 78% (11/14) of Normal Controls showed a REA.

Analysis of asymmetry scores on the nonverbal Complex Tone task revealed no differences between the groups, $F(2,37) = 1.13$, NS. However, in comparison to Normal Controls (LEA $M = -.32$), the Nonanxious Depression group showed essentially no ear advantage ($M = .001$), and the Anxious Depression group showed an intermediate LEA ($M = -.16$). Nine of 14 Normal Controls (64%), 8 of 14 (57%) Anxious Depressives, and 5 of 12 (42%) Nonanxious Depressives showed a LEA on this nonverbal task. Pearson chi-square analysis revealed no differences between the groups in the number of subjects showing an LEA, $X^2(2) = 1.38$, NS.

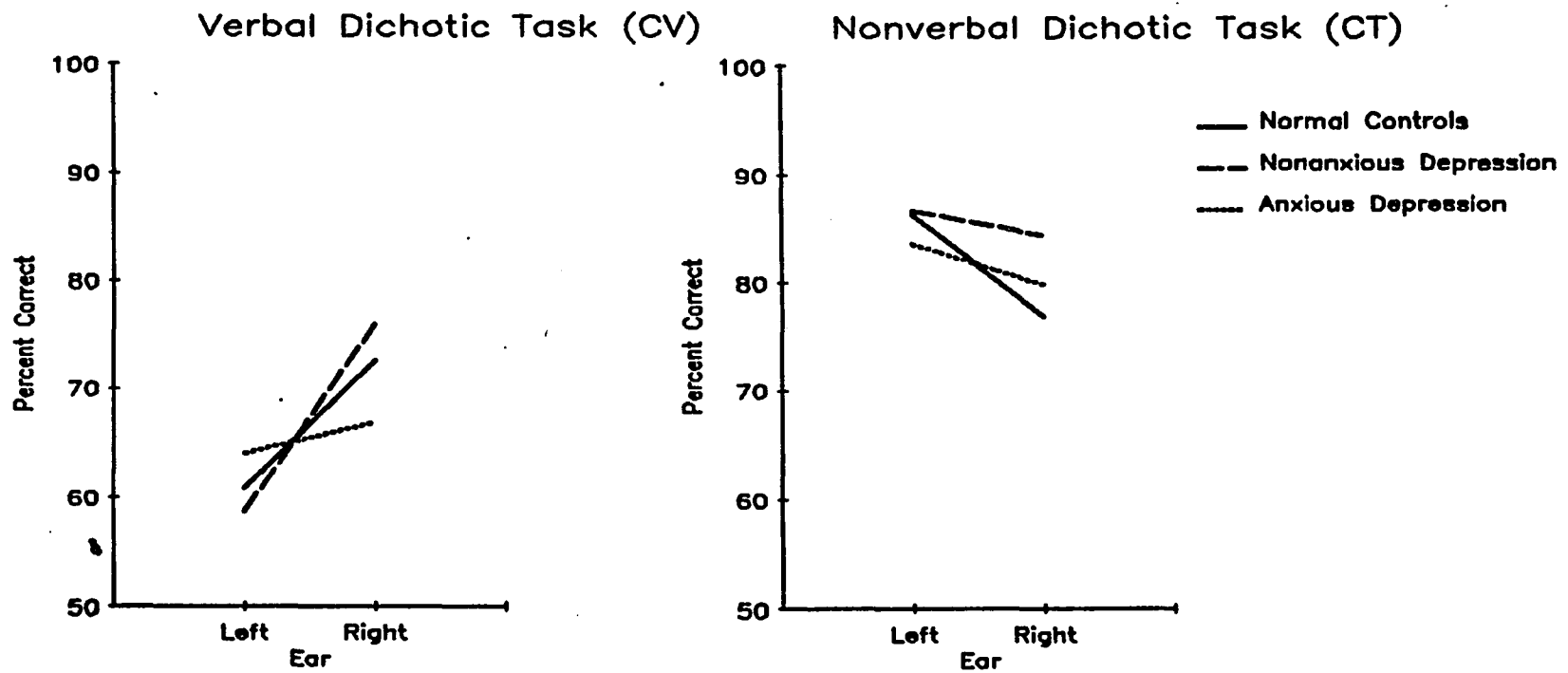
b. Accuracy Scores. In order to further delineate these group differences, accuracy (percent correct) scores were calculated and subjected to a Group X Task X Ear ANOVA (see

Figure 7). The main effect of Task was highly significant, indicating that overall, subjects tended to perform better on the nonverbal (CT; $\bar{M} = 82.72\%$) than on the verbal (CV; $\bar{M} = 66.38\%$) dichotic task, $F(1,37) = 61.65$, $p < .00001$. The Task X Ear interaction was also highly significant, $F(1,37) = 24.94$, $p < .00001$, indicating differences in ear advantages for the two tasks. The 3-way interaction was not significant, $F(1,37) = 2.21$, NS.

However, analysis of performance on the verbal CV task revealed a Group X Ear interaction, which approached significance, $F(2,37) = 2.66$, $p = .08$. Individual two-tailed t-tests indicated that both the Normal Control group ($\bar{M} = 11.69\%$) [$t(13) = 3.62$, $p < .01$] and the Nonanxious Depression group ($\bar{M} = 17.26\%$) [$t(11) = 3.41$, $p < .01$] showed a significant right ear advantage (REA) on this verbal task. In contrast, the Anxious Depression group did not show a significant REA ($\bar{M} = 2.86\%$) [$t(13) = .59$, NS]. Further one-way analyses revealed no differences between the groups for either left or right ear performance.

Analysis of performance on the nonverbal Complex Tone Task revealed an overall left ear advantage ($\bar{M} = 5.33\%$), $F(1,37) = 5.01$, $p < .05$, but no Group X Ear interaction, $F(2,37) = .92$, NS. While the left ear advantage ($\bar{M} = 9.52\%$) of the Normal Control group showed only a trend toward significance, $t(13) = -1.94$, $p = .07$, it tended to be larger than that of the Anxious ($\bar{M} = 3.74\%$) and Nonanxious ($\bar{M} =$

Figure 7. Mean accuracy scores for dichotic listening tasks.



2.28%) Depression groups. There were no significant differences between the groups in either left or right ear performance levels on this task.

4. Relationship between EMG and Perceptual Asymmetry

To determine the relationship between facial muscle activity and auditory perceptual asymmetries, Pearson product-moment correlations were conducted between these variables for each experimental group (see Table 4). There did not appear to be any systematic relationship between these variables. For Anxious Depressed subjects, no correlation was found between their abnormal performance on the verbal dichotic task, and facial muscle activity. However increased corrugator activity (especially during the happy [$p < .05$] and sad [$p < .01$] conditions) was significantly associated with a larger left ear advantage (i.e., more negative asymmetry scores) on the nonverbal complex tone task.

B. Comparison of Depressed Subjects vs. Normal Controls

To test the second hypothesis, that depression in general is characterized by right hemisphere dysfunction, all depressed subjects were compared to normal controls on measures of expression (facial EMG activity) and appreciation (objective ratings) of facial emotion and the dichotic listening tasks.

1. Expression of Facial Emotion

a. Facial EMG Magnitude. To determine if depressed

Table 4. Pearson Correlations for Facial EMG and Dichotic Listening Measures.

CORRUGATOR									
Affect Condition	<u>Normal Controls</u>			<u>Anxious Depression</u>			<u>Nonanxious Depression</u>		
	N	H	S	N	H	S	N	H	S
Verbal (CV) Task									
Right Ear %	-.43	-.36	-.11	.35	.40	.25	.43	.44	.15
Left Ear %	-.19	.02	.21	.04	.15	.26	.26	.27	.20
e	-.39	-.52	-.29	.18	.17	-.04	.18	.21	.00
Nonverbal (CT) Task									
Right Ear %	-.32	-.27	-.05	.25	.08	.04	-.15	-.09	-.09
Left Ear %	-.23	-.22	.31	.19	.10	.18	-.06	.02	-.01
e	-.08	.23	-.37	-.44	-.60*	-.78**	.07	.06	-.04
ZYGOMATIC									
Affect Condition	<u>Normal Controls</u>			<u>Anxious Depression</u>			<u>Nonanxious Depression</u>		
	N	H	S	N	H	S	N	H	S
Verbal (CV) Task									
Right Ear %	.20	.34	.05	-.08	-.14	.15	-.44	-.42	-.41
Left Ear %	.49	.46	.32	.18	.40	.26	-.18	-.52	-.53
e	-.25	-.10	-.30	-.25	-.43	-.09	-.20	-.03	.03
Nonverbal (CT) Task									
Right Ear %	.41	.35	.27	.14	.01	.10	.37	.10	.06
Left Ear %	.35	.36	.11	.03	.26	.33	.33	.17	.17
e	.18	.19	.29	-.22	-.43	-.45	.19	-.06	-.08

*: $p < .05$

** : $p < .01$

subjects as a group differed from Normal Controls in facial EMG patterns, a Group X Muscle X Affective Condition ANCOVA was run. Only the Muscle X Affective Condition interaction was significant, $F(2,103) = 36.27$, $p < .00001$, indicating that the corrugator and zygomatic muscles exhibited different patterns of magnitude in response to affective faces. The 3-way interaction was not significant, indicating that these patterns did not differ for the two groups, $F(2,103) = 1.90$, NS.

Separate Group X Affective Condition ANCOVAs were employed for the corrugator and zygomatic muscles, to further determine the EMG pattern specific to each muscle.

Corrugator. For the corrugator, the main effect of Affective Condition was highly significant, $F(2,103) = 25.62$, $p < .00001$. The Group X Affective Condition interaction was not significant, $F(2,103) = 2.36$, $p = .10$. However, post-hoc Tukey HSD analyses of the individual groups revealed that while both the normal controls and the depressed patients showed reduced corrugator activity for happy faces in comparison to neutral and sad faces ($p < .05$), the normal controls showed equal corrugator magnitudes for neutral and sad faces, but depressed subjects showed greater corrugator magnitude for sad faces than for neutral faces ($p < .05$). Additionally, depressed patients showed significantly greater corrugator EMG ($M = 1.73$, $SD = .35$) magnitude during exposure to happy faces than normal

controls ($M = 1.60$; $SD = .25$), $F(1,51) = 4.56$, $p < .04$.

Zygomatic. Analysis of zygomatic muscle responses revealed a highly significant main effect of Affective Condition, $F(2,103) = 18.70$, $p < .00001$. Neither the main effect of Group nor the interaction were significant, indicating no overall magnitude or pattern differences between the two groups for this muscle. Post-hoc analyses showed that both depressed patients and normal controls exhibited greater zygomatic activity in response to pictures of happy faces than to pictures of sad and neutral faces ($p < .01$). Zygomatic responses to pictures of sad and neutral faces were not significantly different.

b. Time Course of Facial EMG. When time course of facial EMG patterns were measured, the "within slide" Group X Muscle X Affective Condition X Time ANCOVA revealed, as above, a significant Muscle X Time interaction, $F(3,155) = 22.32$, $p < .0001$. There were no differences in "within slide" time course between the depressed and nondepressed groups or across affective conditions.

The "within block" Group X Muscle X Affective Condition X Time ANCOVA revealed, as above, a significant Muscle X Time interaction, $F(1,52) = 6.73$, $p = .01$, indicating "within block" time course differences for the corrugator and zygomatic muscles. There were no differences in "within block" time course between groups or across affective conditions.

2. Appreciation of Facial Emotion

To assess whether the depressed and nondepressed groups differed in their ability to judge the valence and intensity of the affective faces, objective ratings of facial emotion were subjected to a Group X Affective Condition ANOVA. The main effect of Affective Condition was highly significant, $F(2,104) = 337, p < .00001$. Post-hoc Tukey HSD tests revealed that overall, subjects rated happy faces as more happy than neutral and sad faces, sad faces as more sad than neutral and happy faces, and neutral faces as less happy than happy faces and less sad than sad faces ($p < .01$). The interaction was not significant, indicating no differences between the depressed and nondepressed groups in their ability to judge the valence and intensity of facial affect.

3. Auditory Perceptual Asymmetry

a. Assymetry (e) Scores. Results of a Group X Task ANOVA revealed a highly significant Task effect, $F(1,36) = 15.85, p < .001$, indicating differences in ear advantage for the two tasks. The Group X Task interaction was not significant, $F(1,36) = 2.06, NS$, indicating no difference in ear advantage between the groups for either task. When analyzed separately, there were no differences in magnitude of REA for the depressed ($\bar{M} = .16$) and control ($\bar{M} = .20$) groups on the verbal CV task, $F(1,39) = .39, NS$. Although depressed patients appeared to show a smaller mean LEA ($\bar{M} = -.08$) on the nonverbal CT task than normal controls ($\bar{M} = -$

.32), this difference was not significant, $F(1,37) = 1.71$, NS.

b. Accuracy Scores. The results of a Group X Task X Ear ANOVA revealed a highly significant Task X Ear interaction, $F(1,38) = 25.00$, $p < .00001$, indicating differences in ear advantages for the two tasks. There were no differences in ear advantage for depressed and normal subjects on the two tasks $F(1,38) = 1.63$, NS. Individual analyses of ear advantage for the verbal CV task indicated that both depressed patients ($M = 9.51\%$) [$F(1,25) = 6.48$, $p < .02$] and normal controls ($M = 11.69\%$) [$F(1,13) = 13.11$, $p < .01$] showed a significant REA on this task. There were no differences between the groups in either left or right ear performance levels.

Overall, subjects showed a significant left ear advantage on the nonverbal CT task ($M = 5.33\%$ overall) $F(1,38) = 6.93$, $p = .01$. However, when analyzed separately, normal controls showed a trend for a significant LEA on this task ($M = 9.52\%$) [$F(1,13) = 3.78$, $p = .07$] and depressed patients did not ($M = 3.07\%$) [$F(1,25) = 1.72$, NS]. There were no differences between the groups in either left or right ear performance on this task.

C. Relationship Between Expressed and Experienced Emotion

Because of the significant differences in facial EMG patterns found when comparing anxious and nonanxious depressed subjects and normal controls, analyses of the

relationship between ratings of experienced affect and EMG measures were performed separately for each of these experimental groups.

1. Immediate Emotion Experience

One secondary hypothesis predicted that the immediate experience of emotion would correspond to patterns of facial muscle activity. In order to determine whether group differences existed in the immediate reported experience of emotion in response to pictures of facial affect, subjective emotion ratings were subjected to a Group X Affective Condition ANOVA. As for the objective ratings, subjective intensity ratings on the "Happy" scale were scored as positive numbers (i.e., +1 to +5 for judgments of low to high intensity of happy affect, respectively), and those on the "Sad" scale were scored as negative numbers (i.e., -1 to -5 for low to high intensity of sad affect, respectively). The results of this analysis can be seen in Figure 8. The main effect of Group was significant, $F(2,51) = 5.10$, $p < .01$, indicating overall differences between the groups in the level of reported experience of emotion. The results of one-way ANOVAs comparing individual means can be seen in Table 5. Patients in the Anxious Depression group rated themselves as significantly sadder than Normal Controls in response to neutral and happy faces, but not to sad faces, $p < .05$. The Nonanxious Depressed group did not differ significantly from the other two experimental groups.

Figure 8. Mean subjective emotion ratings (immediate experience).

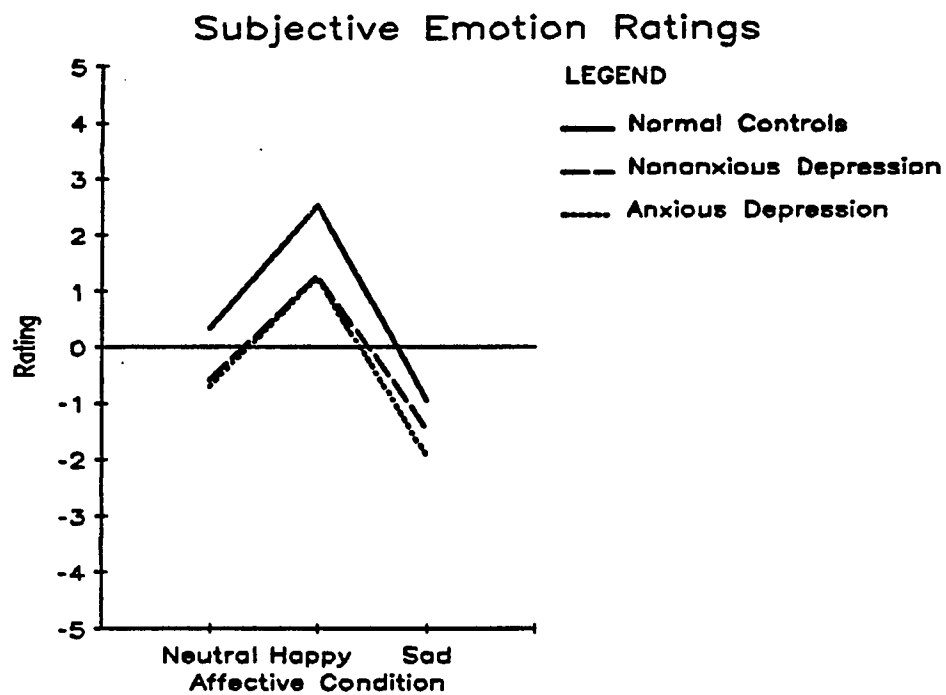


Table 5. Mean Subjective Facial Emotion Ratings.

		<u>Normal Controls</u>	<u>Anxious Depression</u>	<u>Nonanxious Depression</u>	p
Affective Condition					
Neutral	$\bar{X}$ (SD)	.31 (1.47)	-.72 (1.45)	-.59 (.90)	<.04 NC> AD
Happy	$\bar{X}$ (SD)	2.52 (.96)	1.22 (2.04)	1.25 (1.70)	<.03 NC> AD
Sad	$\bar{X}$ (SD)	-.96 (1.64)	-1.91 (1.47)	-1.49 (1.62)	NS

Visual inspection of Figure 8 suggested that both patient groups rated themselves as sadder overall than the normal control group. In addition, when all depressed patients were compared to controls, the patients rated themselves as significantly sadder ($\bar{M} = -.37$) than controls ($\bar{M} = .62$), $F(1,52) = 10.10$, $p < .01$.

The Affective Condition effect was highly significant, $F(2,102) = 88.27$, $p < .00001$, indicating overall differences in reported experience of emotion as a function of the affective quality of the slides. Post-hoc Tukey HSD tests revealed that all subjects reported feeling more happy in response to happy faces than in response to neutral and sad faces, they reported feeling more sad in response to sad faces than neutral and happy faces, and reported feeling less happy and less sad in response to neutral faces than in response to happy and sad faces, respectively ($p < .01$). The Group X Affective Condition interaction was not significant, $F(4,102) = .43$, indicating no differences in the pattern (or "reactivity") of reported emotion experienced by the experimental groups in response to pictures of facial affect.

The pattern of reported experience of emotion (see Figure 8) did parallel the normal pattern of corrugator and zygomatic response (see Figure 2), suggesting an association between the facial EMG response and the reported experience of emotion. Pearson correlation coefficients (two-tail) were

computed in order to determine whether significant associations existed between facial EMG and reported emotion for each subject group.

Corrugator. The correlation coefficients for corrugator muscle activity and reported emotion during exposure to the affective slides can be seen in Table 6. Normal controls showed a significant correlation between reduced corrugator activity and increased reported experience of happiness during exposure to happy faces, $r = -.57$, $p < .01$. In contrast, Nonanxious Depressed patients showed a significant correlation between increased corrugator activity and increased reported experience of sadness, $r = -.55$, $p < .05$, in response to sad faces [the correlation has a negative sign in this case because scores on the "Sad" rating scale were given negative values]. Anxious Depressed patients, on the other hand, showed no significant associations between reported experience of emotion and corrugator activity in response to any of the affective slide conditions.

Zygomatic. Correlation coefficients for reported experience of emotion and zygomatic muscle activity can be seen in Table 7. Both Normal Controls ($r = .46$) and Nonanxious Depressed patients ($r = .56$) showed a significant correlation between increased zygomatic activity and reported experience of happiness in response to happy faces, $p < .05$. In contrast, Anxious Depressed patients showed no significant associations between reported experience of

Table 6. Pearson Correlations for Corrugator EMG and Self-Report Measures of Emotion.

Affect Condition	<u>Normal Controls</u>			<u>Anxious Depression</u>			<u>Nonanxious Depression</u>		
	N	H	S	N	H	S	N	H	S
Immediate									
Neutral	-.32			-.36			-.11		
Happy		-.57**			-.34			.07	
Sad			-.20			-.28			-.55*
State Emotion									
Depression	.07	.15	.05	.20	.02	.10	.05	.04	-.01
Anxiety	.13	-.02	.10	.40	.03	.16	.04	.08	.04
Trait Emotion									
Depression	-.19	-.33	-.15	.07	-.34	-.22	.23	.26	.47t
Anxiety	.15	.19	.26	.24	.07	.06	-.16	-.09	-.07
Anhedonia									
Physical	.01	.15	-.10	-.19	-.10	-.06	-.05	.07	.03
Social	.04	.05	-.21	-.17	.03	.03	-.28	-.36	-.18

t: $p < .10$

*: $p < .05$

** : $p < .01$

Table 7. Pearson Correlations for Zygomatic EMG and Self-Report Measures of Emotion.

Affect Condition	<u>Normal Controls</u>			<u>Anxious Depression</u>			<u>Nonanxious Depression</u>		
	N	H	S	N	H	S	N	H	S
Immediate									
Neutral	.14			-.35			-.14		
Happy		.46*			-.03			.56*	
Sad			.01			-.11			-.19
State Emotion									
Depression	.17	-.08	.23	-.10	-.24	-.40	-.13	-.51*	-.22
Anxiety	.22	-.28	-.03	.26	.09	.02	.04	-.37	-.06
Trait Emotion									
Depression	.23	.13	-.17	-.11	-.30	-.40	.63**	.40	.72**
Anxiety	.32	-.18	.08	-.20	-.25	-.11	.31	.09	.19
Anhedonia									
Physical	.25	.00	.07	-.39	-.50*	-.36	.48t	.51*	.54*
Social	.26	.18	.45*	-.40	-.34	-.05	.29	.45t	.24

t: $p < .10$
 *: $p < .05$
 **: $p < .01$

emotion and zygomatic activity in response to any of the affective slide conditions.

2. State vs. Trait Emotion

Another secondary hypothesis suggested that if facial muscle output was "intimately" associated with the experience of emotion, then because of temporal proximity, immediate measures of reported affect should be most closely correlated with muscle activity, and that state measures of negative affect (e.g., depression and anxiety) should be more closely correlated with muscle activity than trait measures of affect. In addition, it was predicted that a measure of social anhedonia would be associated with reduced zygomatic activity. Mean self-report emotion ratings for each experimental group can be seen in Table 8. Both groups of depressed subjects reported higher levels of anxiety, depression, and anhedonia than normal controls ($p < .001$). There were no significant mean differences between Anxious and Nonanxious Depressed subjects on these rating scales.

Corrugator. Pearson correlation coefficients for self-report measures of affect and corrugator muscle activity can be seen in Table 6. There were no significant correlations between corrugator muscle activity and self-report measures of state and trait emotion for any of the experimental groups.

Zygomatic. Correlations between state and trait measures of emotion and zygomatic muscle activity can be

Table 8. Mean Self-Report Emotion Ratings for Subject Groups.

		<u>Normal Controls</u>	<u>Anxious Depression</u>	<u>Nonanxious Depression</u>	<u>p*</u>
State Emotion					
Depression	$\bar{X}$ (SD)	5.00 (4.15)	19.65 (7.12)	15.35 (7.02)	< .00001 NC< AD = ND
Anxiety	$\bar{X}$ (SD)	29.50 (6.06)	52.18 (12.41)	45.76 (9.48)	< .0001 NC< AD = ND
Trait Emotion					
Depression	$\bar{X}$ (SD)	.40 (.82)	6.59 (2.58)	5.76 (2.49)	< .0001 NC< AD = ND
Anxiety	$\bar{X}$ (SD)	32.40 (7.60)	58.81 (6.36)	58.73 (8.64)	< .0001 NC< AD = ND
Anhedonia					
Physical	$\bar{X}$ (SD)	6.65 (4.52)	14.00 (8.85)	12.12 (7.06)	< .006 NC< AD = ND
Social	$\bar{X}$ (SD)	5.05 (4.06)	17.19 (6.79)	13.37 (6.32)	< .0001 NC< AD = ND

* based on F, one-way ANOVA, with post-hoc Tukey HSD.

seen in Table 7. For Normal Controls, there were no significant associations between state or trait depression and anxiety and zygomatic activity. Normal Controls showed a significant association between high Social Anhedonia and increased zygomatic activity in response to sad faces ($r = .45$, $p < .05$). There were no significant associations between zygomatic activity and Physical Anhedonia for the Normal Control group. Anxious Depressed subjects showed a significant association between high Physical Anhedonia scores and reduced zygomatic activity in response to happy faces ($r = -.50$, $p < .05$). There was no significant association between Social Anhedonia and zygomatic activity for Anxious Depressives. Nonanxious Depressives showed a number of significant correlations between measures of self-reported affect and zygomatic activity. High state depression (DACL) was associated with reduced zygomatic muscle activity in response to happy faces ($r = -.51$, $p < .05$). In contrast, while measures of trait "dysphoric affect" (e.g., anxiety, depression, anhedonia) tended to be associated with reduced zygomatic activity in the Anxious Depression group, trait depression and anhedonia tended to be associated with increased zygomatic activity in the Nonanxious Depression group, regardless of affective condition. Trait depression was significantly associated with increased zygomatic activity in response to pictures of neutral ($r = .63$, $p < .01$) and sad ($r = .72$, $p < .01$) faces,

but not significantly so for happy faces ($r = .40$, NS). Trait anxiety was not significantly associated with zygomatic activity. There was a significant association between high Physical Anhedonia scores and increased zygomatic activity in response to happy ($r = .51$, $p < .05$) and sad ($r = .54$, $p < .05$) faces.

3. Changes Following Antidepressant Treatment Response

It was also predicted that if the corrugator muscle is the effector of "negative" affect, and the zygomatic muscle is the effector of "positive" affect, that the resolution of depression following treatment should be accompanied by a reduction in corrugator activity and an increase in zygomatic activity. Changes in facial EMG muscle patterns were assessed for a subgroup of depressed subjects who responded to treatment, and all normal controls. While 12 of the 17 subjects in each of the patient groups responded to treatment, only 8 Anxious and 9 Nonanxious Depressed patients were available for retest. The depressed subjects were retested after 6 to 12 weeks of treatment with variety of antidepressants: 11 (6 anxious; 5 nonanxious) were taking Prozac (fluoxetine), 3 (2 anxious; 1 nonanxious) were taking Tofranil (imipramine), 2 (nonanxious) were taking a placebo, and 1 (nonanxious) was in a double-blind drug study and was taking either a placebo or Gepirone (an experimental antidepressant).

There were no differences in age, education, or

handedness laterality quotients for the experimental groups at retest (see Table 9). At retest, both the Anxious Depressed [$t(7) = 4.62$; $p < .01$] and Nonanxious Depressed [$t(7) = 4.53$; $p < .01$] subjects showed significant reductions in severity of depression, as measured by the BDI. Although their scores decreased, neither group of depressed subjects showed significant reductions on the state measures of depression and anxiety [depression: AD: $t(7) = 1.59$; ND: $t(8) = 2.06$; anxiety: AD: $t(7) = 1.32$; ND: $t(8) = 1.47$]. Anxious Depressed subjects had significantly higher scores on the BDI and on the state anxiety and depression scales than Normal Controls at retest. Nonanxious Depressed subjects' scores did not differ from either of the other groups.

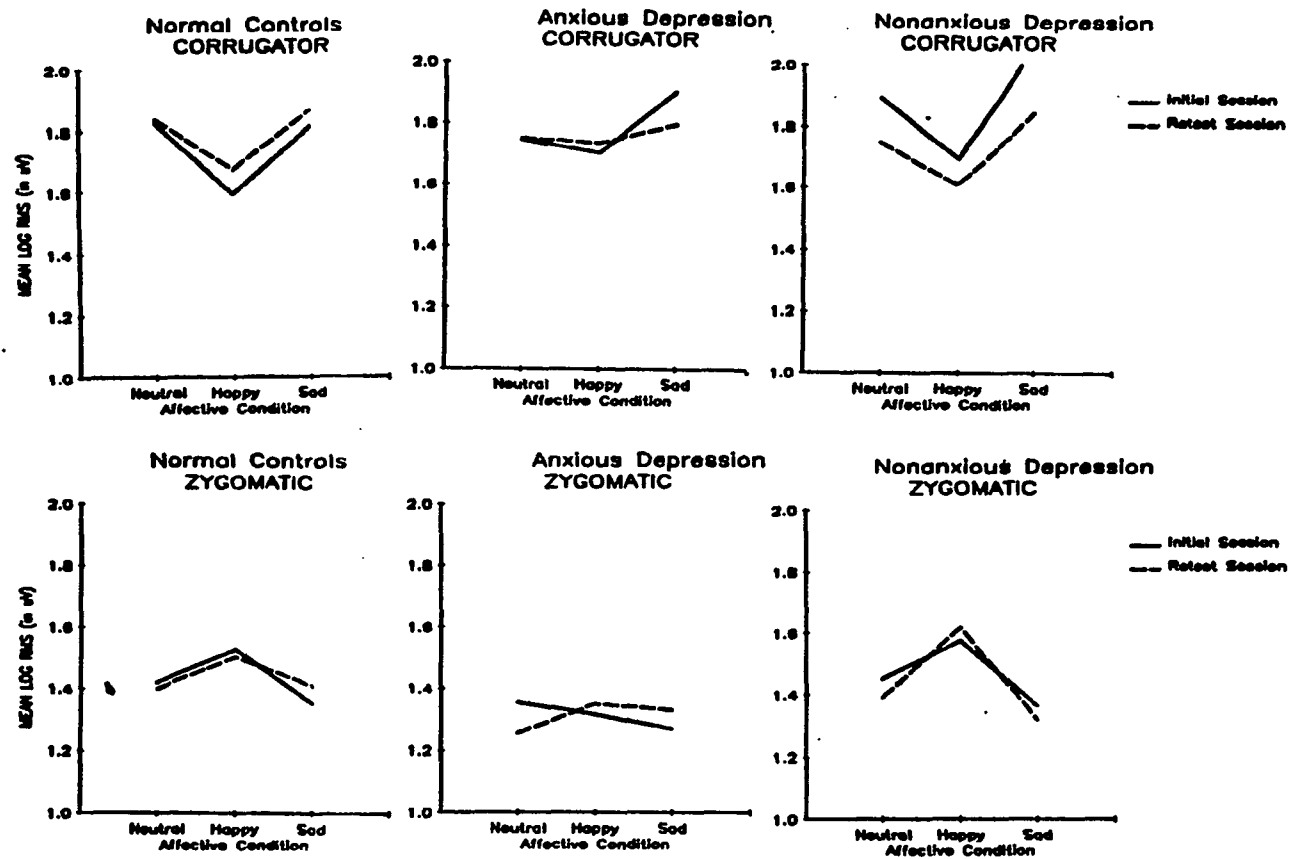
Scores of facial muscle activity were subjected to a Session X Group X Muscle X Affective Condition ANCOVA (see Figure 9). The Session X Affective Condition interaction was significant, $F(2,67) = 3.42$, $p < .05$, indicating that overall facial muscle patterns varied across sessions. The Group X Muscle X Affective Condition interaction was also significant, $F(4,67) = 3.10$, $p < .05$, indicating that facial muscle patterns among the groups were significantly different. However, the Session X Group X Muscle X Affective Condition interaction was not significant, $F(4,67) = .48$, NS, indicating no changes in the muscle patterns of the groups across sessions.

Table 9. Subject Characteristics and Self-Report Measures of Emotion at Retest.

		<u>Normal Controls</u>	<u>Anxious Depression</u>	<u>Nonanxious Depression</u>	<u>p*</u>
N		20	8	9	
Age	<u>X</u>	32.00	39.50	32.33	NS
	(<u>SD</u>)	(10.04)	(10.80)	(10.57)	
Education	<u>X</u>	17.00	15.00	15.56	NS
	(<u>SD</u>)	(3.20)	(2.14)	(1.74)	
LQ	<u>X</u>	55.25	84.79	56.05	NS
	(<u>SD</u>)	(59.94)	(18.46)	(63.85)	
Beck Depression Inventory (BDI)					
Pre	<u>X</u>	1.90	28.75	21.38	< .00001
	(<u>SD</u>)	(1.92)	(11.62)	(9.84)	NC< AD = ND
Post	<u>X</u>	2.95	13.12	6.75	< .01
	(<u>SD</u>)	(5.11)	(10.29)	(7.59)	NC < AD
State Depression (DACL)					
Pre	<u>X</u>	5.00	20.75	14.55	< .00001
	(<u>SD</u>)	(4.15)	(6.30)	(5.20)	NC< ND < AD
Post	<u>X</u>	6.00	14.00	9.22	< .05
	(<u>SD</u>)	(4.48)	(10.61)	(6.34)	NC < AD
State Anxiety (STAI)					
Pre	<u>X</u>	29.50	52.25	43.44	< .00001
	(<u>SD</u>)	(6.06)	(11.55)	(9.32)	NC< AD = ND
Post	<u>X</u>	28.70	42.25	35.78	< .05
	(<u>SD</u>)	(9.68)	(18.95)	(13.37)	NC < AD

*based on F, one-way ANOVA; post hoc Tukey HSD

Figure 9. Comparison of changes in corrugator and zygomatic muscle activity following successful treatment of depression.



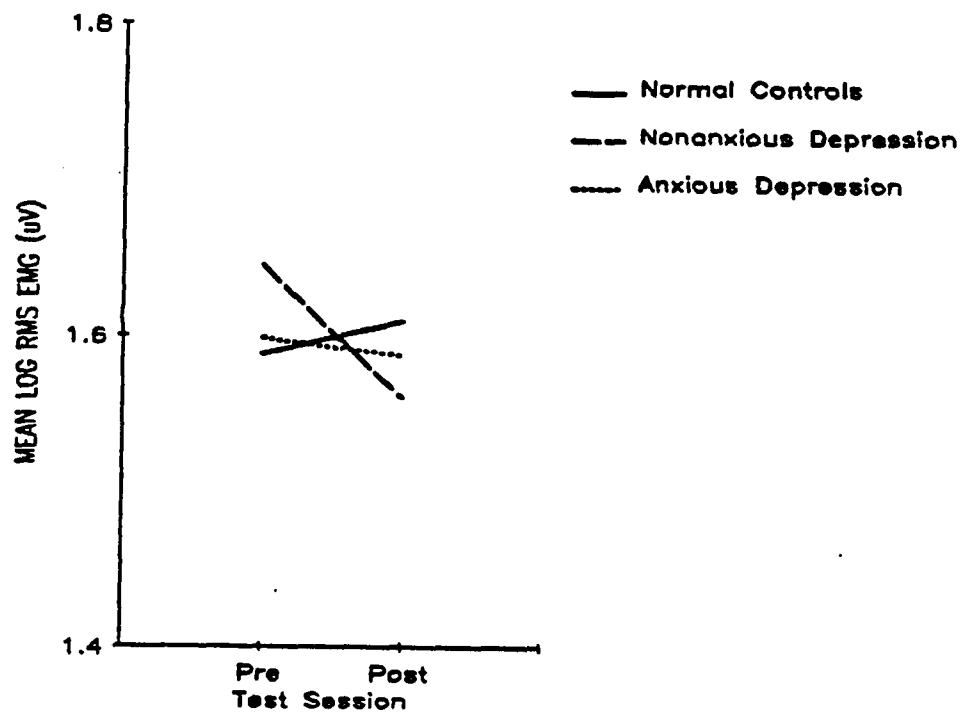
A significant Session X Group interaction was found, $F(2,33) = 4.03$, $p < .05$ (see Figure 10). Post-hoc analyses revealed that Nonanxious Depressed subjects showed an overall reduction in muscle activity following successful treatment ($p < .05$), while Anxious Depressed subjects did not.

Separate Session X Group X Affective Condition ANCOVAs were performed for each muscle to further delineate the differences in muscle activity at retest.

Corrugator. Only the main effect of Affective condition was significant, $F(2,67) = 26.38$, $p < .0001$, indicating a significant "emotional" facial muscle pattern. The Session X Group interaction approached significance, $F(2,33) = 2.89$, $p < .07$, indicating a trend for differences in corrugator activity across sessions for the groups. On visual inspection of Figure 9, the Nonanxious Depression group appeared to show a reduction in corrugator activity, while the Anxious Depression and Normal Control group did not. The Group X Affective Condition interaction also approached significance, $F(4,67) = 2.10$, $p < .10$, indicating a trend for the subject groups to show differential corrugator muscle patterns. The Session X Group X Affective Condition interaction was not significant, however, indicating no significant changes in corrugator muscle patterns at retest, $F(4,67) = .84$, NS.

Session X Affective Condition ANCOVAs were performed

Figure 10. Changes in overall muscle activity following successful treatment of depression.



for each experimental group to further assess pre- and post-treatment muscle patterns.

Anxious Depressed patients showed a significant effect of Affective Condition for corrugator activity, $F(2,13) = 4.99$, $p < .05$, which did not change following successful treatment, $F(2,13) = 1.27$, NS. However, visual inspection of Figure 9 reveals a "flattening" of muscle activity, similar to that seen for the entire group initially. Post-hoc Tukey HSD tests revealed that initially, these treatment responders showed significantly greater corrugator activity in response to sad faces than in response to happy faces, $p < .05$, yet at retest there were no significant differences in corrugator activity in response to affective faces.

Nonanxious Depressed patients showed a significant effect of Affective Condition for corrugator activity, $F(2,15) = 13.45$, $p < .001$. Post-hoc Tukey HSD tests revealed that initially, these treatment-responsive patients showed significantly lower corrugator activity in response to pictures of happy faces than in response to sad ($p < .01$) and neutral ($p < .05$) faces. At retest, they showed significantly less corrugator activity in response to happy than sad faces ($p < .05$). Although the pattern of activity did not change following successful treatment, $F(2,15) = .54$, NS, corrugator activity in general was reduced at retest, ($\bar{M} = 1.88$ vs. $\bar{M} = 1.73$), $F(1,7) = 6.23$, $p < .05$.

Normal Controls showed a significant effect of

Affective Condition for corrugator activity, $F(2,37) = 16.17$, $p < .0001$, which did not change at retest, $F(2,37) = .25$, NS. Post-hoc Tukey HSD tests revealed that Normal Controls exhibited significantly less corrugator activity in response to pictures of happy faces than in response to neutral ($p < .05$) and sad ($p < .01$) faces.

Zygomatic. When zygomatic activity was analyzed separately, a significant Group X Affective Condition interaction indicated differential zygomatic muscle patterns among the groups, $F(4,67) = 2.59$, $p < .05$, which did not change at retest [Session X Group X Affective Condition: $F(4,67) = 1.57$, $p = \text{NS}$]. The Session X Affective Condition interaction approached significance, $F(2,67) = 2.79$, $p < .07$, indicating a trend for overall pattern changes at retest.

When each group was analyzed separately, the Anxious Depressed group showed the same "flat" (i.e., nonreactive) pattern of zygomatic muscle activity, both before and after successful antidepressant treatment, $F(2,13) = .06$, NS.

The Nonanxious Depressed group showed a significant reactive pattern of zygomatic muscle activity, $F(2,15) = 9.31$, $p < .01$, with greater activity in response to happy than sad faces ($p < .05$). Following successful treatment, this pattern showed a trend toward heightened responsivity to happy faces, $F(2,15) = 2.71$, $p < .10$.

The Normal Control group showed a significant reactive

pattern of zygomatic muscle activity, $F(2,37) = 10.52$, $p < .001$, with greater activity in response to happy faces than to sad or neutral faces. This pattern did not change at retest, $F(2,37) = 2.02$, NS.

Because the individual patient groups were rather small, all depressed patients were compared to Normal Controls. Results of a Session X Group X Affective Condition ANCOVA for corrugator activity revealed only a strong main effect of Affective Condition, $F(2,69) = 31.02$, $p < .0001$, and a trend for Session X Group interaction, $F(1,34) = 4.12$, $p = .05$. While depressed patients showed a strong trend for overall reduction in corrugator activity ($\bar{M} = 1.83$ vs. $\bar{M} = 1.74$) following successful treatment, Normal Controls showed a slight increase in corrugator magnitude ($\bar{M} = 1.74$ vs. $\bar{M} = 1.78$) at retest. There were no differences in pattern of corrugator activity between the groups, $F(2,69) = 1.98$, NS, or across test sessions, $F(2,69) = .97$, NS.

For zygomatic muscle activity, an overall main effect of Affective condition was found, $F(2,69) = 17.24$, $p < .0001$. There were no differences in pattern of activity between the groups, $F(2,69) = .12$, NS, or across test sessions, $F(2,69) = 1.98$, NS.

a. Patterns which may predict treatment response. Of interest was that initially, the Anxious Depression group had shown an abnormal, flattened pattern of corrugator activity in response to the affective faces, yet the group

of treatment responders had shown significantly greater corrugator activity in response to sad than happy faces. This suggested that pretreatment patterns of muscle activity may predict treatment response. There were 12 responders and 5 nonresponders for each of the depressive subtypes. Pretreatment corrugator and zygomatic magnitude were subjected to separate Subtype X Response (Responder vs. Nonresponder) X Affective Condition ANCOVAs (see Figure 11).

Corrugator. A significant Subtype X Affective Condition interaction was found, $F(2,59) = 4.02$, $p < .05$, indicating differential corrugator activity patterns for the two depressive subtypes. The three-way interaction was not significant, $F(2,59) = 1.35$, indicating that the differences in corrugator patterns between the depressive subtypes did not differ for treatment responders and nonresponders.

However, the Response X Affective Condition interaction approached significance, $F(2,59) = 2.52$, $p < .10$, indicating a trend for overall corrugator pattern differences between treatment responders and nonresponders (see Figure 12). Post-hoc Tukey tests revealed that responders showed a more "reactive" pattern of corrugator response, particularly greater activity to sad faces than to happy and neutral faces ($p < .01$), while nonresponders showed a lack of corrugator reactivity to affective faces.

Because visual inspection of Figure 11 indicated pattern differences between the depressive subtypes,

Figure 11. Preliminary comparison of pretreatment facial EMG patterns in treatment responders and nonresponders.

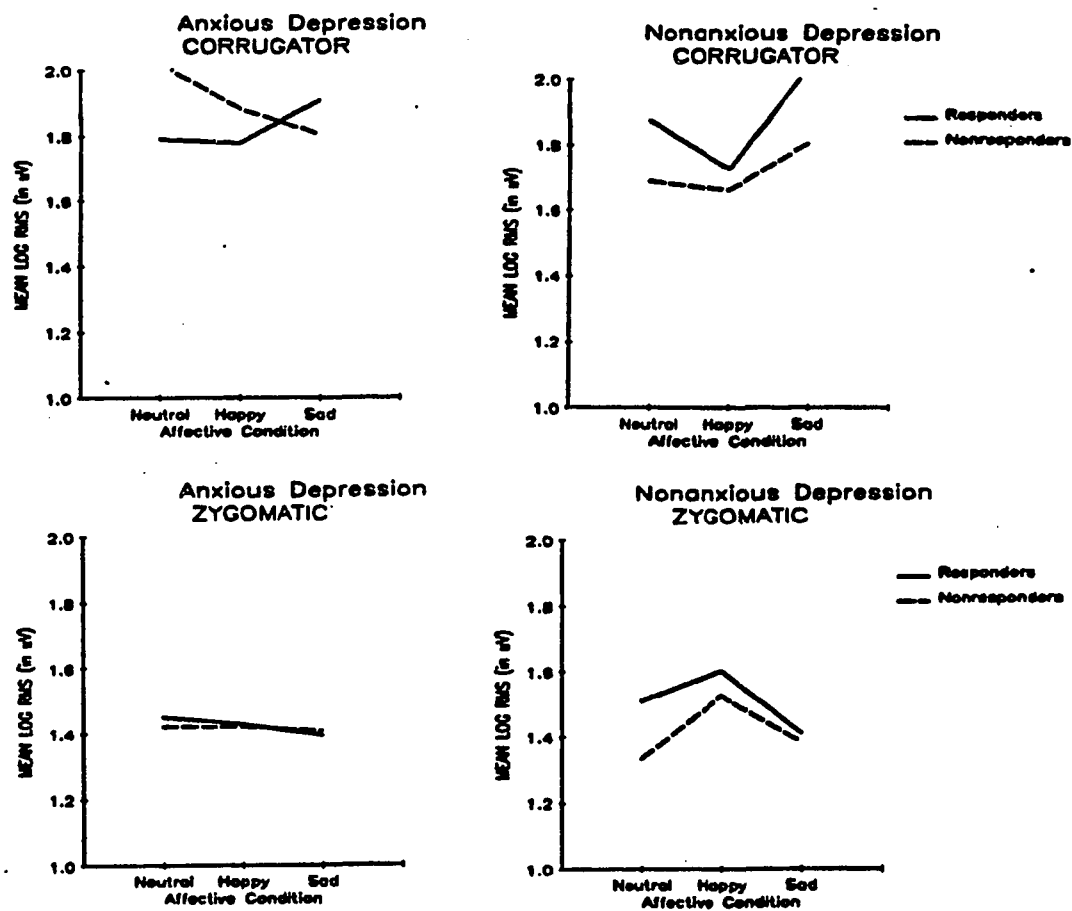
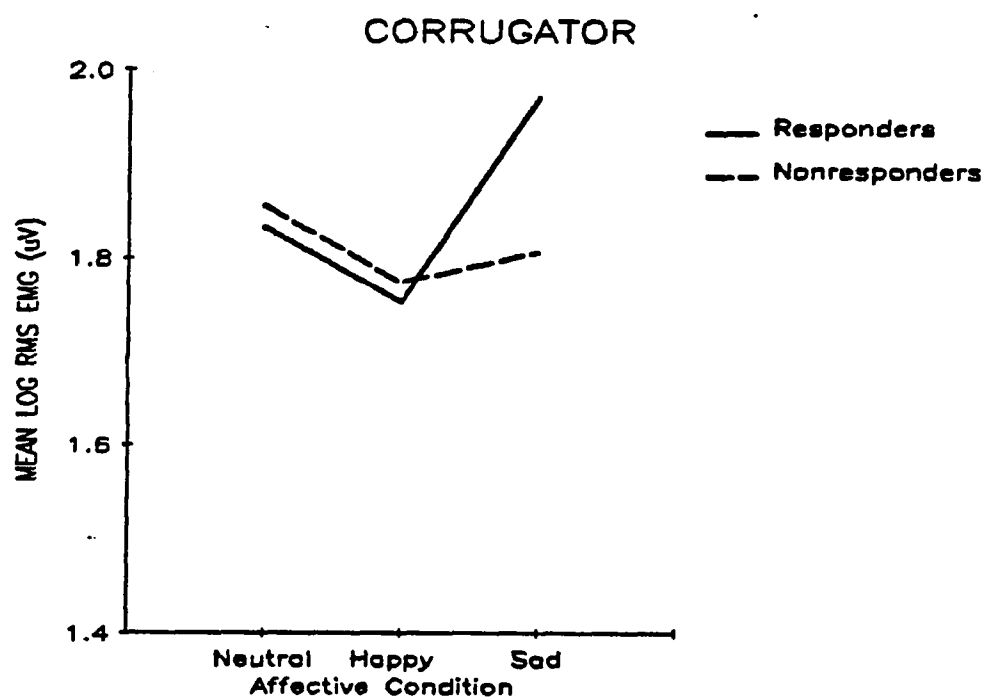


Figure 12. Corrugator muscle activity pattern for treatment responders and nonresponders.



separate Response X Affective Condition ANCOVAs were run for each depressive subtype. Although subject groups were small and require cautious interpretation, some interesting differences resulted. For Anxious Depressed subjects, treatment responders showed a significantly different corrugator muscle pattern than nonresponders, $F(2,29) = 4.60$, $p < .01$. Post-hoc tests revealed that responders showed a marginally elevated corrugator response to sad faces ($p = .05$) than to happy faces. Anxious Depressed nonresponders showed significantly elevated corrugator response to neutral faces in comparison to treatment responders ($p < .01$), as well as in comparison to happy ($p < .05$) and sad faces ($p < .01$).

In general, the Nonanxious Depressed subjects showed significantly greater corrugator activity in response to sad faces than to happy ($p < .01$) and neutral ($p < .05$) faces. There were no differences in magnitude or pattern of corrugator activity for treatment responders vs. nonresponders, $F(2,29) = .18$, NS.

Zygomatic. For zygomatic muscle activity, only the Subtype X Affective Condition interaction was significant, $F(2,59) = 3.62$, $p < .05$, indicating significant differences in facial EMG patterns for the Anxious and Nonanxious Depressed groups (see Figure 11). The three-way interaction was not significant, $F(2,59) = .60$, NS, indicating no differences in magnitude or pattern of zygomatic activity

for treatment responders vs. nonresponders.

When the depressive subtypes were analyzed separately, both Anxious Depressed treatment responders and nonresponders showed a lack of "reactive" zygomatic activity patterns in response to the affective faces, $F(2,29) = .34$, NS. The Nonanxious Depressed group showed significantly greater zygomatic activity in response to happy faces, than to neutral and sad faces, $F(2,29) = 10.15$, $p < .001$. There were no differences in zygomatic activity patterns for treatment responders vs. nonresponders.

Discussion

A. Anxious and Nonanxious Depression

This study found evidence of psychophysiological and neuropsychological differences between anxious and nonanxious depressive subtypes. Anxious depressed subjects showed a striking lack of facial muscle reactivity in response to pictures of happy faces, a lack of normal association between facial muscle activity and self-reported experience of happiness in response to happy faces, and a lack of normal ear advantage on both a verbal and nonverbal dichotic listening task. In contrast, nonanxious depressed subjects showed heightened corrugator muscle activity to sad faces, an abnormally strong association between corrugator activity and self-reported experience of sadness in response to sad faces, and a lack of normal left ear advantage on the nonverbal dichotic task.

The primary hypothesis of greater right hemisphere dysfunction in anxious depression, compared to nonanxious depression, was not supported by the current experiment. Rather, it is suggested that anxious depression (particularly the presence of panic anxiety in "atypical" depression) may be characterized by greater left hemisphere dysfunction (as evidenced by a lack of right ear advantage on a verbal dichotic task) than nonanxious atypical depression. This left hemisphere involvement may be associated with a lack of physiological responsivity to

positive affective stimuli, and a subsequent dissociation of the expression and experience of positive affect. There appears to be a relationship between reduced reactivity to happy faces in anxious depression and physical anhedonia, which may also be related to left hemisphere involvement. It is also suggested that nonanxious atypical depression may be characterized by mild right hemisphere dysfunction (as evidenced by a lack of left ear advantage on a nonverbal dichotic task), a tendency to "overreact" physiologically to sad faces, and an abnormal association between expressed and experienced sadness in response to sad faces.

1. Facial EMG

In comparison to normal controls, anxious depressed subjects exhibited a distinct "lack of reactivity" in both corrugator and zygomatic muscle response to happy faces, whereas nonanxious depressed subjects exhibited evidence of "overreactivity" of corrugator response to sad faces.

The pattern of corrugator activity exhibited by the nonanxious depressed subjects is in accord with the hypotheses of Beck (1963, 1967, 1971), who states that depression may involve an overemphasis on negative stimuli. Depressed individuals have been found to be physiologically overreactive to noxious physical stimulation (Gatchel, McKinney, & Koebernick, 1977; Lewinsohn, Lobitz, & Wilson, 1973; Zuckerman, Persky, & Curtis, 1968), to negative as compared to positive feedback (Golin, Hartman, Klatt, Munz,

& Wolfgang, 1977), and to negative and even neutral affective statements (Suarez, Crowe, & Adams, 1978).

Beck also cites a corresponding lack of normal responsivity to positive stimuli in depressed patients, which is seen in the pattern exhibited by the anxious depressed subjects. Data relevant to the affective impact of positive events on depressed persons are as yet inconclusive, but suggest that depressed persons evidence average to deficient physiological responses to positive feedback (Golin, et al., 1977), and a deficient response to positive affective imagery (Schwartz, et al., 1976a). In addition, depressed subjects have been found to underestimate rates of positive feedback (Nelson & Craighead, 1977), and to show a dissociation between the expression and experience of pleasure (e.g., Brown, 1981; 1982).

Lang, Bradley and Cuthbert (1990) have recently postulated a theoretical model of emotion that may account for the physiological responses of depressed patients to emotional stimuli. Based on research using the startle-probe methodology, these authors suggest that reflexes with the same valence as an ongoing emotional state are augmented, while mismatched reflexes are inhibited. This may explain depressed patients' particular physiological sensitivity to aversive situations, and may provide an explanation of sorts for anhedonia--the inability to

experience pleasure from pleasurable stimuli.

The results of this study, however, suggest that physiological hypersensitivity to negative affective stimuli and physiological hyposensitivity to positive stimuli, may characterize different subtypes of depression--the nonanxious and anxious depressive subtypes, respectively.

Of interest is that anxiety (e.g., Fridlund et al., 1986) and agitated depression have been associated with increased physiological activity, while retarded depression has been associated with reduced physiological activity (Lader, 1975). The current findings appear contrary to this notion, in that anxious depressed subjects did not exhibit increased facial EMG magnitude, and self-report measures of anxiety were not associated with increases in EMG activity. However, the anxious depressed patients in the current study had mainly panic anxiety, which may lead to physiological overarousal only during discrete panic states (see Taylor & Arnow, 1988).

The pattern exhibited by the anxious depressed subjects in the current study is in accord with studies suggesting a particular impairment in the expression of positive emotion in depression (Jaeger, et al., 1986; Levin, et al., 1985), and is similar to that found by several research groups who assessed facial EMG in depressed subjects. Schwartz et al. (1976a) found that depressed subjects (considered "depressed" by ratings on the Zung scale) failed to show the

normal decrease in corrugator activity in response to "happy" affective imagery, and showed increased corrugator activity in response to neutral, "typical day" imagery. This was explained as evidence of a "miniature sad facial expression" in depression. Additionally, two studies have found a "lack of emotional reactivity" in depressed subjects, as evidenced by their lack of distinct facial EMG patterns. Greden et al. (1986) found depressed patients (with Major Depressive Disorder) in general to be less "reactive" to conditions of affective imagery, and found that only endogenously depressed subjects differed from normal controls in EMG magnitude. These endogenous depressed patients showed increased corrugator activity (particularly in response to sad imagery) and increased zygomatic activity (at rest and in response to sad and "typical day" imagery) compared to normal controls. Wexler et al. (1989) found similar results for depressed patients in response to pictures of facial expressions. Depressed patients failed to show the normal "reactive" facial EMG patterns, and showed overall increases in magnitude of corrugator and zygomatic muscle activity. The results of the current study expand these findings, and suggest that, at least among atypically depressed patients, those with a concomitant anxiety disorder may be particularly likely to show a lack of facial muscle reactivity, specifically in response to positive affective stimuli. Of note, a tonic increase in "dysphoric"

facial muscle activity might be expected to result in a reduced EMG response to "positive" affective stimuli. Although this possibility was considered for the anxious depressed subjects in the current study, the fact that the experimental groups showed no difference in baseline EMG magnitude would argue against such a notion.

There are two possible reasons why the anxious depressed and not the nonanxious depressed group may have appeared more like the depressed patients in previous studies. First, most of the patients in the current study had "atypical" symptom features, the core feature of which is mood reactivity (Liebowitz, et al., 1984). These patients tend to react to both positive and negative environmental stimuli. They can be cheered, at least temporarily, by positive events (e.g., they can enjoy a good meal) and often become depressed following a negative environmental event (e.g., the breakup of a romantic relationship). It may be that nonanxious atypical depressives show relatively normal or enhanced reactivity to affective stimuli, while anxious atypical depressives respond more like "typical" or endogenous depressives. Anxious depressed patients in general have been found to have symptoms previously associated with endogenicity (see Clayton, 1987). In addition, anhedonia (loss of interest or pleasure) is thought to be particularly associated with endogenous or "endogenomorphic" depression (Klein, 1974), suicidal

ideation, and dexamethasone nonsuppression in depression (Oei, Verhoeven, Westenberg, Zwart, & Van Ree, 1990).

Anhedonia is of particular interest here because the anxious depression group showed a specific lack of responsivity to pictures of happy faces, which was significantly associated with a measure of physical anhedonia. Second, it may be that depressed subjects in previous studies had significant anxiety symptoms or anxiety disorders, which might explain the similarity of their findings to those of the anxious depressed patients in this study. None of the studies mention excluding patients with anxiety symptoms. Although several patients in the Wexler et al. study were taking anxiolytic medications at the time of EMG measurement, it was not stated whether patients met psychiatric diagnostic criteria for anxiety disorders.

2. Relation of Facial EMG to Experienced Emotion

Both groups of depressed subjects reported feeling happier in response to happy faces and sadder in response to sad faces. This affective reactivity might be expected in these atypically depressed subjects, whose mood is generally reactive to environmental stimuli. However, further support for the anxious/nonanxious subtyping distinction was found in the differential abnormalities of association between facial muscle activity and reported experienced emotion for the two groups. Anxious depressed subjects showed a lack of normal association between experienced happiness and facial

muscle activity in response to happy faces, e.g., they reported feeling happier but showed a lack of normal physiological response to happy faces. In contrast, nonanxious depressed subjects showed an abnormal degree of association between experienced sadness and corrugator activity in response to sad faces, a finding that is in accord with this groups' corrugator "overreactivity" to sad faces. Furthermore, a measure of physical anhedonia was associated with reduced zygomatic muscle activity in response to happy faces for the anxious depressed subtype, yet anhedonia and a measure of trait depression were associated with increased zygomatic activity in nonanxious depressed subjects.

These abnormalities of association between facial EMG activity and reported experienced emotion in depression support similar findings in previous studies (Brown, 1981; 1982; Brown, Schwartz, & Sweeney, 1978; Brown, Sweeney, & Schwartz, 1979). Brown (1982) has explained the abnormal association of expressed and experienced sadness in depression in terms of cognitive psychology, and proposes that depressed patients may not possess appropriate screening mechanisms to protect them from negative or painful stimuli. Brown et al. (1979) have found a lack of normal association between self-perception of pleasure and emission of observable pleasurable behaviors in schizophrenic and depressed patients. These researchers

suggest that two separate, interactive pleasure "systems" which mediate the expression and experience of pleasure may be dysfunctional in depression. In the current study, both depressive subtypes failed to show normal expression-experience associations, but their patterns of abnormality differed. The abnormal association of facial muscle activity and experienced sadness in nonanxious depression may implicate deficient screening mechanisms for negative affective stimuli in this subtype, while the lack of association of expressed and experienced happiness in anxious depression may implicate a dissociation of pleasure systems in this depressive subtype.

In the present study, several factors may have influenced these expression-experience relationships, and they must therefore be interpreted cautiously. First, subjects were limited to a choice between only two emotions, happy or sad, for which to describe their subjective feelings. It is possible that, given a wider range of emotion choices, resultant expression-experience relationships may have differed. Second, subjective ratings of emotion always followed objective ratings. Although subjects were specifically instructed to try to be accurate as to how they actually felt during exposure to each slide, it is possible that the preceding objective emotion ratings may have influenced their subjective emotion ratings. Finally, Buck (1980) has stated that intersubject

relationships between facial expression and experienced emotion may be quite different from intrasubject relationships between these variables. Future studies which include a wider range of emotion choices by which to describe experienced emotion, a randomized method for rating objective and subjective emotions (e.g., by giving a separate exposure for one rating type), and which examine the frequency with which facial EMG and affective experience change in the expected direction for individuals, will likely strengthen interpretations regarding expression-experience associations, and may offer new insight into their relationship to symptom and personality features.

3. Appreciation of Facial Emotion

The prediction that depressed patients would show abnormalities in their ability to appreciate facial expressions was not supported by the data. Neither group of depressed patients showed differences in their ability to judge the emotional valence (happy vs. sad) or intensity of affective faces, in comparison to normal controls. This may be related to the "mood reactive" quality of their atypical depressions. The ability of such atypically depressed subjects to accurately judge the affective quality of face-to-face social stimuli may explain their reactivity of mood, particularly in response to interpersonal relationships (see Liebowitz, et al., 1984).

However, it is also possible that the task used was not

of sufficient sensitivity to measure what may be more subtle deficiencies of facial emotion appreciation in depression. Cooley and Nowicki (1989) reported that depressed patients had slower reaction times than normal controls in discriminating facial expressions. In addition, Feinberg, et al. (1986) found hospitalized depressed patients to show deficits in labeling facial expressions under tachistoscopic presentation, and yet, Walker, McGuire and Bettles (1984) found no differences when stimuli were presented for longer time periods. In the current study, subjects judged only two emotions, "happy" and "sad", during a 4 second stimulus presentation. It may be that under more difficult cognitive processing requirements, e.g., a wider range of emotion choices, shorter presentation time, and/or reaction time paradigm, differences in the appreciation of facial emotion would have emerged. Therefore, from the current results, it is impossible to determine whether anxious and nonanxious depressed patients differ in their ability to appreciate facial emotion, or to process faces in general. Future studies should include additional measures of visuospatial perception to determine whether visuospatial or facial emotional processing abnormalities characterize these depressive subtypes.

4. Perceptual Asymmetry

Anxious and nonanxious depressed subjects also showed differences in perceptual asymmetries measured by dichotic

listening tasks. Both groups showed a lack of significant left ear advantage (LEA) on the nonverbal dichotic task, in accord with prior studies suggesting right hemisphere dysfunction in depression (e.g., Berger-Gross, et al., 1985; Bruder, et al., 1981; Johnson & Crockett, 1982). However, anxious depressed subjects also showed a striking lack of right ear advantage (REA) on the verbal dichotic listening task, a finding which may support more left hemisphere dysfunction in this subtype. Nonanxious depressed subjects showed a significantly larger REA (over 14%) on the verbal task than anxious depressed subjects.

In terms of dichotic listening laterality, the performance of nonanxious depressives suggests mild right hemisphere dysfunction. Although they were not significantly different from normal controls on either dichotic task, nonanxious depressives' lack of LEA on the nonverbal dichotic task, and slightly larger REA (over 5% compared to normal controls) on the verbal task, may suggest a tendency for right hemisphere dysfunction in this subtype. In contrast, anxious depressives' additional lack of REA on the verbal task, may implicate more left hemisphere (or bilateral) dysfunction in this depressive subtype.

The possibility of left hemisphere involvement in anxious depression is in accord with hypotheses which implicate left hemisphere dysfunction in anxiety (e.g., Tucker, 1981), in sadness, depression, or reduced "positive"

affect (e.g., Henriques & Davidson, 1990), and a recent study of affective disturbance following brain lesions, which associated left cortical lesions with anxious depression (Robinson & Starkstein, 1991).

Findings of studies assessing perceptual asymmetries of depressed patients on verbal dichotic listening tasks have been inconsistent, and may have been affected by severity, subtype, and symptom features (see Bruder, 1988). Some studies have found depressed patients to show a lack of REA on verbal dichotic listening tasks (e.g., in severely depressed patients prior to undergoing electroconvulsive treatment) [Moscovitch, Strauss, & Olds, 1981], which was reported to improve following successful treatment of depression (Johnson & Crockett, 1982; Moscovitch et al., 1981). This lack of REA has been variously interpreted as a decline in left hemisphere function (Wale & Carr, 1990), a "strongly primed right hemisphere" (Moscovitch, et al., 1981), abnormalities in interhemispheric inhibition via the corpus callosum, or abnormal lateral attentional bias or cognitive strategy allocation (Johnson & Crockett, 1982).

The lack of REA shown by anxious depressed subjects on the verbal dichotic task does not appear to be due to a "strongly primed right hemisphere" or an abnormal lateral attentional bias. An abnormally primed right hemisphere would have resulted in both a lack of REA on the verbal task and a larger than normal LEA on the nonverbal task, due

primarily to enhanced left ear performance. An abnormal attentional bias favoring the right hemisphere would have resulted in the same response pattern. Clearly, the anxious depressed subjects in this study tended to show reduced or no lateralization on both dichotic tasks, and this was not due specifically to enhanced unilateral ear performance. Such performance is more consistent with bilateral hemisphere dysfunction, or perhaps abnormal interhemispheric inhibition via the corpus callosum, both of which might result in less lateralized processing for both a verbal and a nonverbal task.

A recent study found symptoms of anxiety in depression to be associated with a normal REA on a verbal task with an attentional component, while symptoms of withdrawal and retardation were associated with a lack of REA (Wale & Carr, 1990). While the results of this study might seem at odds with the lack of REA for the anxious depressed subjects in the current study, it is important to note that "anxiety" in depressed patients may be quite a different phenomenon than "panic anxiety" in depression, which primarily characterized the anxious depressed subjects in this study. There is evidence that the comorbidity of panic and depression may represent a unique affective syndrome (see Pauls & DiBenedetto, 1987).

5. Theoretical Implications

Although there is strong support for right hemisphere

dominance of emotion, there is also some evidence which suggests differential hemispheric specialization for emotion as a function of valence (e.g., "positive" or "negative"). This has been termed the "valence" hypothesis. This hypothesis suggests that negative emotion is primarily associated with the right hemisphere, and positive emotion is associated with the left or both hemispheres (Dimond & Farrington, 1977; Sackeim, Greenberg, Weiman, Gur, Hungerbuhler, & Geschwind, 1982; Tucker, 1981). Physiological studies of heart rate (Dimond & Farrington, 1977), EMG (Schwartz et al., 1979), and lateral eye movements (Ahern & Schwartz, 1979) have suggested that the right hemisphere may be more engaged by negative emotions and the left hemisphere may be more engaged by positive emotions. From EEG studies, right frontal areas are suggested to be involved in negative emotions, while left frontal regions are thought to be involved in positive emotions (see Davidson, 1984, 1985, 1987). Additionally, two variants of the valence hypothesis have been proposed. Borod and Koff (1984) reviewed studies assessing the effect of valence on facial asymmetries, and suggested right hemisphere mediation of negative affect and bilateral mediation of positive affect (i.e., negative expressions are more left-faced, positive expressions are more symmetrical). Other researchers have suggested right hemisphere mediation of emotional perception and valence-lateralized emotional

expression (Davidson, 1985).

Davidson (1985) and his colleagues (e.g., Davidson & Tomarken, 1989; Henriques & Davidson, 1990), have suggested that differential hemispheric frontal activation may characterize persons prone to certain affective states or psychopathology. They suggest that individuals with left frontal hypoactivation may be prone to impairment in "approach" behaviors (e.g., reduced positive affect) and those with right frontal hyperactivation may be prone to increased "withdrawal" behaviors (e.g., increased negative affect). Furthermore, Tucker et al. (1981) have suggested that in depression, increased activation in the frontal areas of the right hemisphere may act to inhibit functioning in the more posterior areas of the same hemisphere, perhaps implicating a link between an abnormal increase in negative affect and a decrease in other right hemisphere functions.

The valence hypothesis may best explain the results of this study. One might speculate that anxious depressed subjects, experiencing left or bilateral hemisphere dysfunction (as evidenced by a lack of REA on the verbal dichotic task and a lack of LEA on the nonverbal dichotic task), do not respond physiologically to happy faces. This is in accord with the notion of left (or bilateral) hemisphere involvement in positive affect. On the other hand, nonanxious depressed subjects, experiencing mild right hemisphere dysfunction (as evidenced by their failure to

show a LEA on the nonverbal dichotic task), overreact physiologically to negative (sad) stimuli. This is in accord with the notion of right hemisphere involvement in negative affect.

Some additional support for left hemisphere involvement in anxious atypical depression comes from findings of similar facial processing and perceptual asymmetry deficits in schizophrenia, a disorder which is thought to involve left hemisphere dysfunction (see Wexler, 1981). Several prior studies have found that schizophrenics also show a lack of REA on verbal dichotic tasks (Colbourn & Lishman, 1979; Johnson & Crockett, 1982). Recent studies have associated this lack of REA with negative symptoms in schizophrenia, suggesting that symptoms such as psychomotor retardation, flat affect and anhedonia may be related to perceptual asymmetries (Carr & Wale, 1988; Wale & Carr, 1988). In addition, schizophrenics have been found to show abnormalities in the perception (e.g., Feinberg, et al., 1986) and expression of (particularly positive) facial emotion (Martin, Borod, Alpert, Brozgold, & Welkowitz, 1990), and to show a lack of normal association between expressed and experienced pleasure (Brown, Sweeney, & Schwartz, 1979). Anxious depressed patients, on the other hand, have been found more likely than nonanxious depressives to experience delusional depressions (Sacchetti, et al., 1987). These similarities may suggest common left

hemisphere involvement in these disorders, which may be related to similar abnormalities in perceptual asymmetry, in the expression of positive facial emotion, and in some symptom features.

B. Depression

In light of the literature suggesting right hemisphere dysfunction in depression, it was hypothesized that depressed patients in general would show abnormalities in the expression and appreciation of facial emotion, and evidence for right hemisphere dysfunction on dichotic listening tasks. In the current study, depressed patients in general showed a lack of left ear advantage on a nonverbal dichotic task, a finding consistent with this hypothesis. However, given the differences found between anxious and nonanxious subtypes in facial EMG activity and on the verbal dichotic task, further interpretation of results for the combined group of depressed subjects is inappropriate. The differences apparent between the groups support the need for distinguishing between anxious and nonanxious subtypes for studies assessing emotional and neuropsychological processes in depression.

C. Facial Muscle Activity and Experience of Emotion

Previous studies found support for a distinction between the corrugator and zygomatic muscles as valence-dependent effectors of emotion (e.g., Fridlund & Izard, 1983). In addition, Dimberg (1989) has stated that the

facial muscles constitute an "emotional output system" and activity in these muscles is "intimately related to the experience of emotion".

The results of this study provided partial support for the hypotheses that the corrugator and zygomatic muscles are effectors of "negative" and "positive" affect, respectively, and that the expression and experience of emotion are closely related. It appears, however, that the extent to which these hypotheses are true depends on the specific affect elicitors, the presence of trait depression, anxiety disorder or panic attacks, and possibly on learned affective-cognitive relationships and skills for regulating expression and subjective experience (Izard, 1990).

1. Response to Pictures of Facial Affect

It appears that for nondepressed, nonanxious control subjects, patterns of facial muscle activity do correspond to reports of ongoing subjective affect in response to pictures of neutral, happy, and sad faces. When normal control subjects were exposed to happy faces, they showed increased zygomatic activity and decreased corrugator activity, both of which were significantly associated with reported feelings of happiness. In comparison to happy faces, when exposed to sad faces, normal control subjects showed increased corrugator activity, reduced zygomatic activity, and reported subjective feelings of sadness.

Although it was thought that exposure to sad faces

would result in increased corrugator activity and/or decreased zygomatic activity as compared to neutral faces, this was not the case for normal controls (although it was the case for nonanxious depressed subjects, who tended to "overreact" physiologically to sad faces). In Pilot Study 1, a gradual increase in corrugator activity had been found in response to sad faces, and it was suggested that examination of time course might yield differences between sad and neutral faces. The data did not support this conclusion. Although significant differences were found between the corrugator and zygomatic muscles for both types of time course measured, time course did not differentiate between the affective faces, or between the experimental groups.

These findings are in contrast with previous studies which have found differential growth and carry-over of corrugator and zygomatic muscle activity depending on induced mood state (Sirota & Schwartz, 1982; Sirota, Schwartz, & Kirsteller, 1987). Such studies have found that both the corrugator EMG activity associated with induced depression and the subjective experience of depression grow over time and carry over into non-imagery rest periods, and induced elation leads to immediate increases in zygomatic activity, which quickly returns to baseline levels during rest periods. These authors speculated that a self-perpetuating cognitive-physiological mechanism may be functioning in depression, resulting in a downward spiral of

mood, and increased length of time to recover from depression in comparison to elation.

There may be several reasons for this failure to show differences in corrugator magnitude and time course in response to sad and neutral faces. First, sad faces may not be of sufficient affective intensity (in comparison to neutral faces) to elicit a differential EMG response in the nondepressed observer. Dimberg (1989) has used pictures of angry faces to elicit the "negative" pattern of corrugator response in normal control subjects. Sad faces were used in the present study in order to parallel previous studies of facial EMG in depression (e.g., Greden, et al., 1986). However, angry faces may evoke a more intense negative affective experience in the observer than sad faces. Borod, Koff, and Buck (1986) suggested that negative emotions, particularly those which are biologically significant (e.g., linked to survival, promoting retreat from a dangerous situation) would require processing systems like those of the right hemisphere (e.g., quick scanning of Gestalt information to expedite a decision to retreat). It is likely, therefore, that angry faces would be of greater biological significance to observers (in terms of retreat and survival) than sad faces, and this may be linked to corrugator activity. Second, neutral faces, on the average, were rated as being slightly sad. Thus, the response to neutral faces may be more "negative" than neutral. Third,

although exposure to affective faces led to valence changes in affective experience, it is unlikely that these faces induced a depressed or elated mood in subjects, which may be required to elicit emotion-dependent time course differences.

2. Self-Report Measures of Emotion

It was predicted that because of the temporal proximity of measures of reported emotion to facial muscle activity, measures of immediate subjective emotional experience would be more closely related to facial muscle activity than measures of state and trait affect. This prediction was partially supported by the data. However, because of the number of correlations executed between measures of facial EMG and self-report measures of emotion, and because of the factors mentioned above which may have influenced measures of experienced emotion, the results of significant associations must be cautiously interpreted.

For normal control subjects, the associations between facial muscle activity and ratings of immediate subjective affect were particularly strong in response to happy faces. Measures of state and trait depression and anxiety were not associated with facial muscle activity. In contrast, the two groups of depressed subjects showed differential associations between facial EMG activity and reports of experienced emotion. In anxious depressed subjects, there was no association between facial muscle activity and

immediate reports of experienced emotion, or state and trait measures of anxiety or depression. In nonanxious depressed patients, who showed an abnormal association between immediate reports of experienced sadness and increased corrugator activity in response to sad faces, an association between state depression and reduced zygomatic activity in response to happy faces was found. Yet a trait measure of depression tended to be associated with increased zygomatic activity, irrespective of affective condition. This is in accord with previous findings that zygomatic muscle activity may be generally increased in depression (e.g., Greden et al., 1986). According to Greden et al. mesolimbic circuitry (affective regulation) and nigrostriatal function (psychomotor regulation) are neurobiologically linked, and dysregulation of mesolimbic networks may persistently "drive" psychomotor networks, resulting in increased muscle activity in depression.

Interestingly, measures of state and trait anxiety did not lead to higher levels of EMG activity, as might have been predicted given an hypothesis of physiological overarousal in anxiety (e.g., Lader, 1980). This may attest to the primary occurrence of panic anxiety in our anxious depression group, which may result in only subtle differences in physiological activity during nonpanic intervals (Taylor & Arnow, 1988).

The results of Pilot Study 1 showed that high trait

depression was correlated with reduced zygomatic activity and less experience of happiness in response to happy faces, and a tendency to rate neutral faces as being more sad. It was thought that this may represent a variant of anhedonia, or an inability to obtain pleasure from pleasurable stimuli. Because of these findings, physical and social anhedonia were assessed in subjects. Facial EMG has been used to measure the affectivity of social interaction processes (Dimberg, 1982; Cacioppo & Petty, 1979), and social interactions have been found to be disordered in depressed patients (e.g., Lewinsohn, 1975). Because human faces were employed as affective stimuli in the current study, it was predicted that measures of social, and not physical, anhedonia would be associated with abnormal patterns of facial EMG activity in response to pictures of affective facial stimuli.

This prediction was partially supported by the data. For anxious depressed subjects, high scores on the Physical Anhedonia scale were associated with the abnormal pattern of reduced zygomatic muscle activity in response to pictures of happy faces. In contrast, for nonanxious depressed subjects, Physical Anhedonia was associated with increased zygomatic activity, irrespective of the affective quality of the faces. This difference further supports the notion of a physiological distinction between these two subtypes.

However, it had been specifically predicted that Social

Anhedonia would correlate with facial EMG, given that the affective stimuli were human faces. Physical, and not social, anhedonia ratings have been found to distinguish good and poor premorbid schizophrenics, and it has been hypothesized that such measures may mark nonschizophrenic persons that are at high risk for psychosis (Chapman & Chapman, 1976). Thus, it may be that the Physical Anhedonia scale measures more severe levels of anhedonia, and may be more likely to reflect a biological defect than the Social Anhedonia scale, which may be affected by social pressure and the subject's desire to present herself in a socially desirable fashion (Chapman & Chapman, 1976). Similarly, the Physical Anhedonia measure may be particularly suited to the physiological nature of the facial EMG task. It appears congruent that subjects who rate themselves as unable to experience physical pleasure, would show a lack of physiological reactivity to positive, or pleasurable, visual stimuli.

3. Depression

The prediction that depressed subjects would show greater corrugator magnitude and lower zygomatic magnitude than normal controls was partially supported by the data. Anxious depressed subjects showed greater corrugator activity and lower zygomatic activity than normal control and nonanxious depressed subjects, respectively, but only in response to happy faces. These findings expand upon previous

studies suggesting a "lack of emotional reactivity" in depression (Greden, et al., 1986; Wexler, 1989), and suggest that emotional nonreactivity may be specific to anxious subtype, to the affective situation at hand (e.g., happy faces), and may be related to anhedonia. Nonanxious depressed patients did not show differences in EMG magnitude for any of the affective conditions in comparison to normal controls.

The prediction that the resolution of depression would be accompanied by a reduction in corrugator activity and an increase in zygomatic muscle activity was also partially supported by the data. Nonanxious, but not anxious, depressed subjects showed a general reduction in both corrugator and zygomatic muscle activity following successful treatment of depression, but no pattern change. The decrease in corrugator activity following alleviation of depression is consistent with the findings of Schwartz et al. (1978), who also found a reduction in corrugator levels following treatment response. The abnormal pattern of EMG in anxious depressed subjects did not normalize following successful treatment. This may indicate that the patterns specific to each subtype represent state-independent (e.g., trait) physiological characteristics.

It should be noted that although the treatment responders were judged to be much improved or very much improved by research psychiatrists, and showed significant

reductions in severity of depression as measured by BDI scores, neither patient group showed significant decreases in scores on the state measures of anxiety and depression. In clinical settings, it is common for the vegetative symptoms of depression to resolve first in treatment, and the subjective feelings of depression to resolve later (Kaplan & Sadock, 1988). This may be why observer judgments of depressed patients' response to treatment may differ from the individual patient's subjective assessment of improvement early in the course of treatment. On the other hand, the discrepancy between patients' self-reported affect and psychiatrists' judgment of improvement may be another aspect of the abnormal associations between expressed and experienced emotion found for these depressed subjects.

In addition, anxious, but not nonanxious, depressed subjects had significantly higher levels of depression and anxiety than normal controls when retested, suggesting that this group may have been experiencing residual symptoms. This is in accord with prior studies which have found anxious depressed individuals to be less responsive to treatment and to experience a more chronic course than nonanxious depressed patients (e.g., Paykel, et al., 1979).

4. Patterns Which May Predict Treatment Response

The results of analyses of initial EMG patterns for treatment responders and nonresponders are tentative, due to small sample sizes. However, these analyses provide

interesting preliminary results on which to base future study. It is suggested that a more distinct (or "reactive") corrugator pattern, particularly elevated corrugator response to sad vs. happy and/or neutral faces, may predict treatment response among depressed individuals. More specifically, among anxious depressives, a somewhat elevated corrugator response to sad faces (e.g., a more "normal" reactive pattern) may predict response to antidepressant medication or placebo, while a highly unusual pattern of elevated corrugator activity in response to neutral faces may predict somatic treatment nonresponse.

These findings, however preliminary, partially support the results of previous studies which have associated increased pretreatment corrugator activity with treatment response (Carney et al., 1981b; Schwartz et al., 1978). However, at least among these atypically depressed individuals, the increase in corrugator muscle activity may be specific to sad faces or other "negative" affective situations. One might speculate that among anxious depressed subjects, the most abnormal corrugator pattern may include those patients who have the poorest prognosis.

5. Theoretical Implications

The results indicate that facial expression and the immediate experience of emotion are related, but perhaps only in specific situations. The notion of a "happy" muscle and a "grief" muscle appears to be too simple a concept--

the nature of the affect elicitor, trait affect, the presence of depression or anxiety disorder, and possibly individual differences in learned cognitive affective structures may differentially affect facial muscle activity patterns and their association with emotional experience.

Izard (1977; 1990; Izard & Malatesta, 1987) has argued that the extent to which an individual regulates his experience of emotions through expression-experience relationships depends on individual stage of development, and personal style and skills for emotion regulation. Izard postulates that in infancy, a closed-loop reflex of emotional response is operating. According to this theoretical position, an internal or external affective stimulus evokes an innate sequence of stimulus evaluation, expression, and subsequent feeling state in the infant. In the adult, through learning processes, the components of the sequence are modified, and complex cognitive-affective structures, not simply the feedback from emotion expression, may be used to regulate subjective feeling states. He hypothesizes that with increasing age, emotions and moods become associated with cognitions, and that such affective-cognitive structures change with age. Such an hypothesis implicates cognitive therapy for the treatment of maladaptive cognitive structures or inappropriate expression-experience relationships.

Although hypotheses of "facial feedback" were not

addressed in this experiment, the anxious atypically depressed subtype provides an interesting group to study from a "facial feedback" point of view. Such patients show a lack of physiological reactivity to affective stimuli, yet report "reactive" patterns of self-experience of emotion. One might speculate that anxious depressed patients are relying solely on cognitive appraisal of environmental stimuli to determine their affective states, while normal controls have the benefit of immediate physiological feedback, particularly in response to happy stimuli, to help determine theirs. On the other hand, the nonanxious depression group may experience an abnormal amount of feedback from sad or negative experiences, in turn enhancing their negative affective states.

D. Clinical Significance

The results of the current study may have particular implications for the social deficits associated with depression because human faces were employed as stimuli. Brown (1982) has raised the concern that social deficits in depression may be due to a disruption of social interchange caused by a consistent dissociation between experienced emotion and behavior communicated to the observer. In the current experiment, spontaneous, covert (i.e., unobservable) facial muscle activity was measured in response to emotional faces (i.e., during a noninteractive task). Although it is unclear how covert facial EMG activity

relates to observable communicative facial emotions, one might speculate that during real social interactions the same abnormal patterns result. If anxious depressed subjects fail to reciprocate positive facial expressions during face to face interactions, and nonanxious depressed subjects are viewed by others as overly "negative", such behavior might create interpersonal negativity and rejection from others (e.g., Coyne, 1976a; 1976b), and/or result in poor social skills (e.g., Lewinsohn, 1974; 1975), thereby complicating depressive symptomatology. Brown (1982) has raised the issue of using biofeedback in the treatment of depressed patients, with the goal of working toward a more normal association of discrepant systems of experience and expression.

These findings suggest that biofeedback or cognitive therapies might be helpful when specifically geared to the abnormalities of expression-experience associations for the individual patient. Indeed, Schwartz et al. (1976b) found that specifically asking depressed subjects to "feel" emotions during affective imagery normalized facial EMG magnitude. Additionally, voluntary unilateral contraction of facial muscles has been found to induce emotion (Schiff & Lamon, 1989). It may be that depressed subjects can be taught strategies for more normal expression-experience relationships. Further research is needed to confirm this suggestion, and to assess the effect of such treatment on mood, social interaction, and neuropsychological function.

E. Conclusions

In conclusion, the results of this study support a psychophysiological and neuropsychological distinction between anxious and nonanxious atypical depressions. It is suggested that anxious atypical depression (particularly panic anxiety in atypical depression) is characterized by greater left hemisphere dysfunction than nonanxious atypical depression. This left hemisphere dysfunction may be associated with a lack of physiological responsivity to pictures of happy faces, and a subsequent dissociation of the expression and experience of emotion in response to happy faces. There appears to be a relationship between this reduced reactivity to happy faces and anhedonia, which may also be related to left hemisphere involvement. It is suggested that nonanxious atypical depression may be characterized by mild right hemisphere dysfunction, a tendency to "overreact" physiologically to sad faces, and an abnormal association between expressed and experienced sadness in response to sad faces.

These results are consistent with the "valence" hypothesis of emotion, which postulates that the processing of positive emotions is mediated predominantly by the left hemisphere, while the processing of negative emotions is subserved by the right hemisphere. Future studies should include other measures of brain function and activation (e.g., EEG, rCBF, clinical neuropsychological tasks) to

confirm the suggestion of differential hemispheric abnormalities in anxious depression. In addition, studies of neuropsychological processing in depression should contrast atypical and "typical" depressive features, nonanxious and anxious depression (including comorbid depression and panic disorder vs. other anxiety disorders), and include groups of patients with primary anxiety disorders (with no concomitant depression) in order to further delineate neuropsychological characteristics specific to anxiety disorders, depressive disorders, and comorbid disorder symptom features.

Some support was provided for the hypotheses that the corrugator and zygomatic muscles are effectors of "negative" and "positive" affect, respectively, and that the expression and experience of emotion are related. It appears, however, that the extent to which these hypotheses are true may depend on the specific affect elicitors, the presence of depression, anxiety disorder or panic attacks, and possibly on learned affective-cognitive relationships and skills for regulating expression and subjective experience. Because the subjective emotion ratings in this study were limited to only two emotions and may have been influenced by previous objective facial emotion ratings, expression-experience relationships must be cautiously interpreted. Future studies should include a wider range of emotion choices, a randomized order of ratings, and examine intrasubject expression-experience relationships to strengthen

interpretations regarding these associations.

It is also suggested that the abnormalities of emotion expression-experience relationships may be related to social deficits in depression, and that the use of biofeedback or cognitive therapies designed to normalize such relationships may be helpful in the treatment of depression. Further study is needed to confirm these hypotheses, and should examine the relationship between covert facial EMG activity and overt communicative facial emotion, as well as their relationship to affective experience and social interaction.

There is preliminary evidence that the specific facial muscle patterns found for the anxious and nonanxious depressive subtypes may be state-independent (trait) markers, and certain patterns may predict treatment response. Further research is needed to confirm these preliminary findings. Future studies should include larger sample sizes, and address the issues of response to specific medications or classes of antidepressant drugs, and specific phenomenological and biological symptom features which may be associated with facial muscle activity patterns and treatment response.

Finally, the differences apparent between the anxious and nonanxious depressed subjects in this study attest to the importance of distinguishing between these subtypes to ensure homogeneous experimental subject groups. Future studies of neuropsychological function in depression should

include careful control procedures for atypical symptomatology and concomitant anxiety disorders. Careful selection of patients for nonanxious, typical symptom features might yield the strongest evidence of right hemisphere dysfunction on verbal and nonverbal dichotic tasks, while atypically depressed individuals with concomittant anxiety (panic) disorder may show more evidence of left hemisphere dysfunction.

Appendix A

Pilot Study 1--EMG Task: Methods

(see Voglmaier & Hakerem, 1989)

1. Purpose

Dimberg (1988) found that pictures of happy and angry facial expressions evoked differential patterns of muscle activity, which corresponded to changes in subjective emotional experience. Because of the literature suggesting a differential time course of the corrugator and zygomatic muscles, and the influence of mood state on these muscles, a pilot study was conducted to a) determine whether our methods were adequate to detect differences in facial EMG patterns in response to pictures of neutral, happy, and sad faces, and their relation to subjective emotional experience; b) examine the time course of the corrugator and zygomatic muscles in response to neutral, happy, and sad faces; and c) examine the relationship of facial EMG patterns and measures of trait affect.

2. Methods

The subjects and methods employed in this study are outlined in the Appendix. There were some differences in the EMG methods as compared to those proposed in the experiment: 1) the block of neutral slides was always given first, to serve as a baseline; 2) the size of the projected image was larger; 3) subject's distance from the image was greater; 4) the data were not log-transformed.

3. Results

The specific results of the study are presented in the appendix, and are summarized as follows:

A. The results of Pilot Study 1 provide further support for the use of pictures of facial affect to elicit characteristic facial muscle patterns which correspond to the subjective experience of differential emotions. The zygomatic and corrugator facial muscles appear to function as an emotional output system, closely related to the experience of happiness and sadness.

B. It is suggested that the "positive" facial muscle pattern associated with the experience of happiness in response to a happy face includes increased zygomatic activity as well as decreased corrugator activity.

C. The "negative" facial muscle pattern associated with the experience of sadness in response to a sad face appears to include a time-dependent increase in corrugator activity.

D. Corrugator muscle activity appears to increase steadily over time in response to a sad face, while zygomatic muscle activity shows a differential trend for variability over time in response to a happy face.

E. There were preliminary indications that facial EMG patterns, subjective experience of emotion, and the ability to judge facial emotion may be differentially affected by positive and negative trait affect. These results may implicate the use of pictures of facial affect in affective

disorders research.

1. High trait depression appears to be associated with the attenuation of both zygomatic muscle activity and subjective experience of happiness in response to happy faces.

2. Conversely, high trait positive affect appears to be associated with the augmentation of both corrugator muscle activity and subjective experience of sadness in response to sad faces.

3. Subjects with high trait depression had a tendency to rate neutral faces as looking sad, while those with high positive affect tended to rate neutral faces as looking happy.

4. Discussion

Based on these findings, the methods used were adequately sensitive to measure indices of spontaneous, covert muscle activity, which may be related to emotional experience. Using these methods, we were able to a) replicate the basic findings of Dimberg's (1988) study, by showing characteristic muscle patterns as well as a correspondance to subjective experience; b) show time course differences of muscles in response to pictures of facial affect which appear similar to those previously found during mood induction procedures (e.g., Sirota & Schwartz, 1982); and c) suggest a relationship between trait mood characteristics, facial muscle patterns, appreciation of

facial affect, and the subjective experience of emotion.

These findings also provide further rationale for using pictures of facial affect to study facial muscle pattern responses in patients with affective disorders. Pictures of facial affect used in this manner appear to provide both an objective method for eliciting spontaneous facial expressions, as well as a measure of appreciation of facial emotion. Given the hypotheses regarding right hemisphere superiority for emotional processes and right hemisphere dysfunction in depression, the relationships suggested by the study between trait affect, muscle activity, appreciation of facial affect, and emotional experience implicate the use of these methods in the neuropsychological investigation of emotion and depression.

Facial electromyography (EMG) in response to facial expressions: Relation to subjective emotional experience and trait affect

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Pictures of happy and angry facial expressions have been found to evoke differential patterns of facial muscle activity which correspond to changes in subjective emotional experience (Dinberg, 1948). The purpose of the present study was to expand on previous findings by examining the magnitude and time course of facial EMG patterns in response to pictures of neutral, happy, and sad faces, and their relation to subjective emotional experience and measures of trait affect. Eleven right-handed female subjects viewed blocks of 12 neutral, 12 happy, and 12 sad facial expressions (Ekman, 1975) for 4 seconds each, with an interstimulus interval of 30 - 45 seconds. Bipolar EMG was recorded from the left corrugator and zygomatic muscle regions. The waveform amplitude was sampled and digitized at a rate of 600/s, and the root mean square (rms) was calculated in consecutive 1 s epochs. Subjects rated each slide for affective intensity and immediate subjective experience of happiness and sadness on 5-point scales. Subject trait characteristics of depression, anxiety, hostility, sensation-seeking and positive affect were measured using the Multiple Affect Adjective Check List-Revised (MAACL-R; Zuckerman & Lubin, 1985).

Sad faces evoked greater mean corrugator activity than happy faces, whereas happy faces evoked greater zygomatic activity, $F(1,10) = 6.83$, $p < .05$, (Figure 1). In comparison to neutral faces, happy faces evoked increased zygomatic activity, $t(10) = 2.62$, $p < .025$, decreased corrugator activity, $t(10) = -3.06$, $p < .01$ (Figure 2), and increased experience of happiness. Sad faces evoked variable increases in corrugator activity, $t(10) = 1.71$, $p = .06$, and increased experience of sadness when compared to neutral faces (Figure 4). There was no difference in zygomatic activity in response to neutral and sad faces. Subjective emotional experience was correlated with objective ratings of stimulus affect for happy ($r = .68$) and neutral ($r = .67$) faces, but not for sad faces. Corrugator activity increased during the 4 second stimulus presentation in response to sad faces, $F(3,30) = 4.05$, $p < .025$, while no time change was seen during happy and neutral stimuli (Figure 3a). Zygomatic activity showed only a trend for change over time in response to happy faces, (Figure 3b). High trait depression was associated with less zygomatic activity ($r = -.61$) and less experience of happiness ($r = -.78$) in response to happy stimuli, and the tendency to rate neutral faces as sad ($r = -.59$). High trait positive affect was associated with increased corrugator activity ($r = .67$) and increased experience of sadness ($r = -.66$) in response to sad faces, and the tendency to rate neutral faces as happy ($r = .63$). These results provide further support for the use of pictures of facial affect to elicit characteristic "positive" and "negative" facial muscle patterns which correspond to differential emotional experience. It is suggested that the "positive" pattern associated with the experience of happiness in response to a happy face includes increased zygomatic activity as well as decreased corrugator activity, while the "negative" pattern associated with the experience of sadness in response to a sad face includes time-dependent increases in corrugator activity. There were also preliminary indications that facial EMG patterns and the experience of emotion in response to facial expressions may be differentially affected by positive and negative trait affect.

Fig. 1 Comparison of Corrugator and Zygomatic Muscle Activity in Response to Pictures of Facial Affect

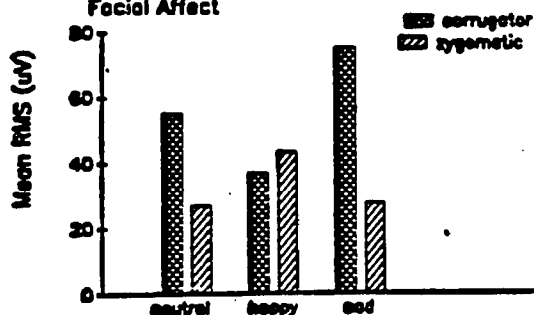


Fig. 3a. TIME COURSE OF CORRUGATOR MUSCLE ACTIVITY

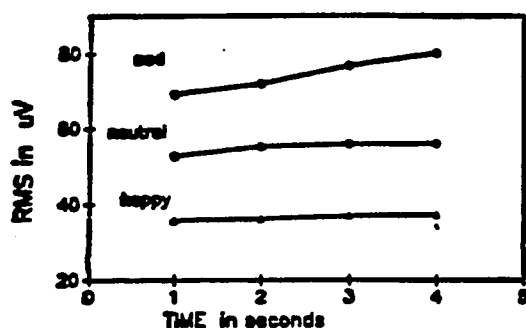


Fig. 2 MEAN MUSCLE ACTIVITY CHANGE FROM NEUTRAL BASELINE

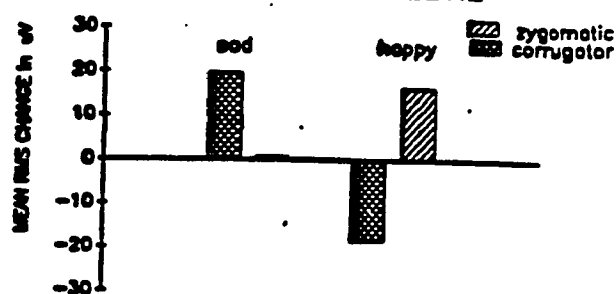


Fig. 3b. TIME COURSE OF ZYGOMATIC MUSCLE ACTIVITY

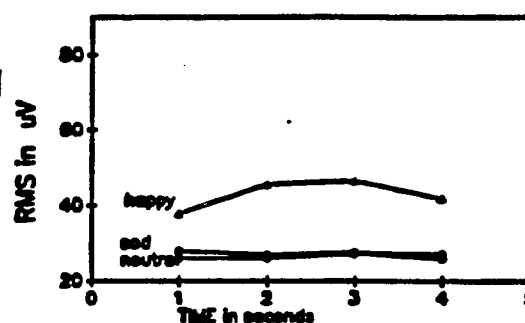
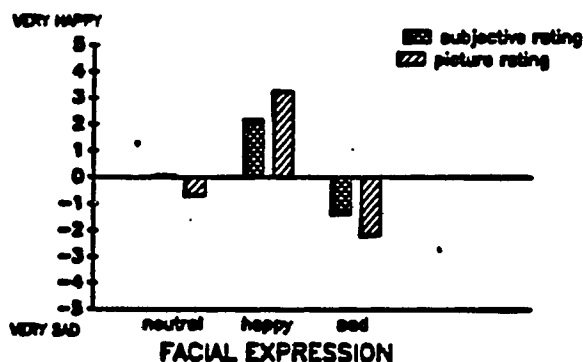


Fig. 4 MEAN EMOTION INTENSITY RATINGS



REFERENCES

- Disberg, U. 1968. Facial electromyography and the experience of emotion. *Journal of Psychophysiology*, 2:277-282.
- Kaan, P. 1975. *Pictures of Facial Affect*. Palo Alto: Consulting Psychologists Press, Inc.
- Zuckerman, M. & Lubin, B. 1985. *MAACL-R: The Multiple Affect Adjective Check List-Revised*. San Diego: Educational and Industrial Testing Service.

Appendix B

Pilot Study 2--EMG Task: Retest Reliability

1. Purpose

The purpose of Pilot Study 2 was: a) to determine if the facial muscle patterns found in Pilot Study 1 were replicable at a new test site, using the methods proposed in the experiment; and b) to examine whether the magnitude and patterns of facial muscle activity were statistically reliable at retest.

2. Methods

Subjects.

The subjects were 18 females (mean age = 32.4; SD = 10.33; range 21 - 56) with no current mood or anxiety disorder, and no history of neurologic or neuromuscular disorder. Subjects were screened for normal or corrected-to-normal vision. All subjects were right handed, except for one, who was ambidextrous. All subjects gave informed consent, and were paid for their participation.

Apparatus, Stimuli, & Procedures.

The methods for the EMG data collection and procedures were the same as will be used in the experiment, described above. All subjects were retested after about 6 to 12 weeks, using the same random order of affective slide blocks (e.g., neutral, happy, sad) as used in the first test session.

Experimental Design.

The pilot study used a 2 X 2 X 3 repeated measures

factorial design. The independent variables were Session (pretest, posttest), Muscle (corrugator, zygomatic), and Affective Condition (neutral, happy, and sad faces). The dependent variable was the mean of log-10 transformed RMS values of EMG activity, measured in microvolt units.

Data Analysis.

To assess whether facial muscle patterns were statistically reliable at retest, an overall Session X Muscle X Affective Condition analysis of covariance (ANCOVA) was conducted (with mean baseline values for each affective condition as the covariates). To determine individual test-retest reliability for each affective condition, Pearson product-moment correlation coefficients (1-tailed) were conducted. An individual Session X Affective Condition ANCOVA was conducted for each muscle, to determine whether the patterns of muscle activity replicated those of Pilot Study 1. T-tests (one-tail) were conducted to determine differences between individual mean EMG values.

3. Results

Test-Retest Reliability.

Results of the overall Session X Muscle X Affective Condition ANCOVA revealed a highly significant Muscle X Emotion interaction (see Figure 1), $F(2,33) = 18.06$; $p < .00001$. There was no effect of Session, and no interaction of Session with any other variables, indicating that both the magnitude and pattern of facial muscle activity evident

at the first test session were replicable after 6 to 12 weeks. Test-retest correlation coefficients (1-tailed) for mean EMG values during the affective conditions ranged from $r = .22$ to $r = .42$ for the corrugator, and from $r = .16$ to $r = .51$ for the zygomatic muscle. These retest reliability coefficients were significant ($p < .05$) for the corrugator muscle during the happy and sad conditions, and for the zygomatic muscle during the neutral condition.

Facial EMG Patterns.

Corrugator Muscle. Results of the Session X Affective Condition ANCOVA for the corrugator muscle revealed a highly significant effect of Affective Condition, $F(2,33) = 16.54$; $p < .00001$. Again, there was no main effect of Session, and no interaction, indicating retest reliability of the muscle activity pattern, $F(2,33) = .57$, NS. Individual t-tests showed that both sad and neutral faces evoked significantly more corrugator activity than happy faces ($p < .05$), in both the pre- and post-test session. There was no significant difference between the corrugator activity evoked by sad and neutral faces.

Zygomatic Muscle. Results of the Session X Affective Condition ANCOVA for the zygomatic muscle also revealed a highly significant effect of Affective Condition, $F(2,33) = 8.61$; $p = .001$. There was no main effect of Session, and no interaction, indicating retest reliability of the zygomatic muscle activity pattern, $F(2,33) = .78$, NS. Individual t-

tests showed that happy faces evoked significantly more zygomatic activity than sad and neutral faces, in both the pre- and post-test session ($p < .05$). There was no significant difference between the zygomatic activity evoked by sad and neutral faces.

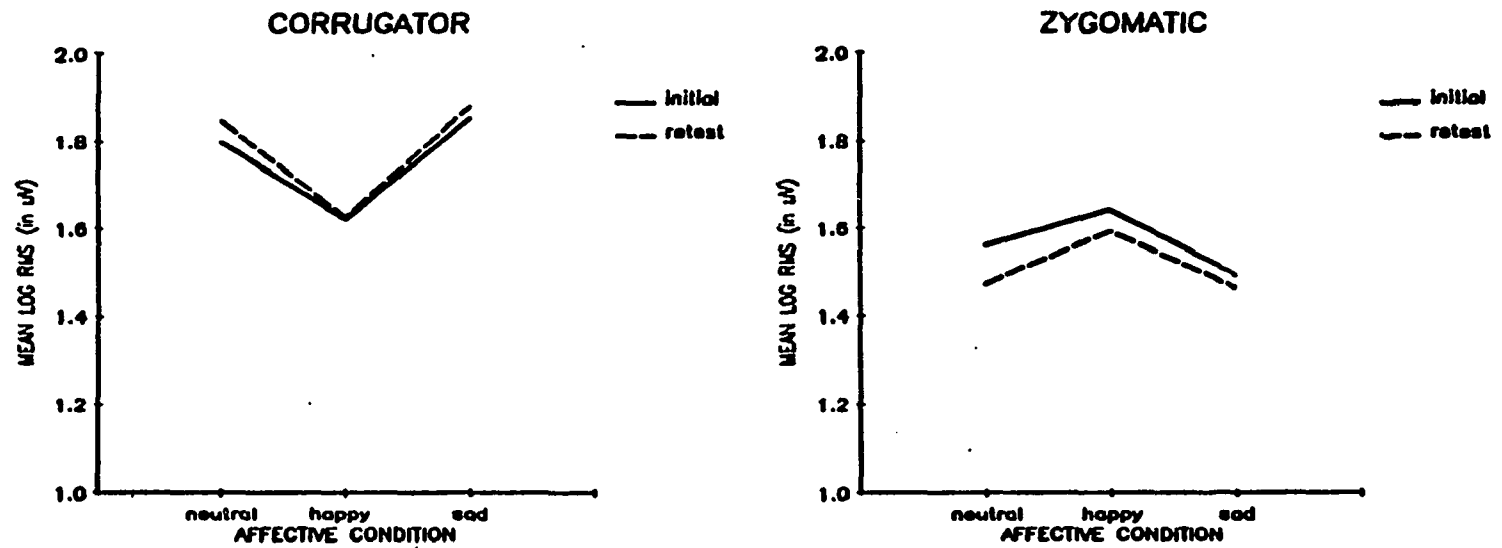
4. Discussion

The results of Pilot Study 2 indicate that the characteristic "positive" and "negative" facial muscle patterns evident in Pilot Study 1 are replicable at the new test site. Specifically, sad faces evoke more corrugator activity than happy faces, while happy faces evoke more zygomatic activity. The finding that, compared to neutral faces, happy faces evoke both an increase in zygomatic activity as well as a decrease in corrugator activity, was also replicated. In Pilot Study 1, sad faces evoked a time-dependent increase in corrugator activity when neutral faces were used as a baseline. In Pilot Study 2, no differences were found in mean corrugator activity evoked by sad and neutral faces. There may be two reasons for this failure to replicate: 1) in Pilot Study 1, neutral faces were always given first as a baseline condition, and the difference may have been associated with an order effect, or 2) the time course of muscle activity was not assessed in Pilot Study 2, and may be necessary to elicit true corrugator effects. Given the literature on the differential "growth" of corrugator activity over time, as well as the results of

Pilot Study 1, the second explanation appears to be more likely, and provides further rationale for assessing time course of muscle activity in the experiment.

The results of Pilot Study 2 also show that these characteristic muscle activity patterns are reliable over time, even after a period of 6 to 12 weeks. This finding provides rationale for assessing possible changes in muscle activity patterns in patients, who respond following 6 to 12 weeks of antidepressant treatment.

Figure 1. Test-Retest Comparison of Facial EMG Patterns During Exposure to Affective Facial Stimuli.



Appendix C

Preliminary Dissertation Data

Facial EMG Patterns in Anxious Depressives, Nonanxious Depressives, and Normal Controls

A. Purpose

A preliminary analysis was conducted in order to determine whether the facial EMG methods were revealing group differences, and to determine the sample size required to detect significant group differences in facial muscle activity patterns.

B. Methods

Subjects.

The subjects included 23 female depressed patients, most of whom had the atypical features described above. Patients were subdivided into two groups:

1) the "Anxious Depression" group (mean age = 37.8) consisted of 11 patients with major depression who also had an anxiety disorder. Ten had atypical disorders, and one had melancholia. Eight of the 11 patients met DSM-IIIr criteria for panic disorder, one patient was given a diagnosis of "probable" panic disorder, and two patients had panic attacks which did not meet the frequency restrictions of DSM-IIIr criteria. Of these 11 patients, one also met criteria for generalized anxiety disorder and social phobia, one for social phobia, one for agoraphobia, and one had a probable diagnosis of generalized anxiety disorder.

2) the "Nonanxious Depression" group (mean age = 34.5) consisted of 12 depressed patients who had no anxiety disorder. Nine patients had major depression, and 3 had dysthymic disorder. Ten of the 12 patients had atypical disorders, and one had probable melancholia.

The Normal Control group (mean age = 32.4) consisted of the same 18 nondepressed, nonanxious females described in Pilot Study 2.

The three groups did not differ in age, $F(2,36) = 1.09$, NS, or education level, $F(2,36) = 1.13$, NS. The handedness laterality quotients of the three groups approached significance, $F(2,36) = 2.93$; $p = .07$. The Normal Control group contained one subject who was ambidextrous, the Anxious Depression group contained 2 left-handed subjects, and the Nonanxious Depression group contained 3 left-handed subjects.

Procedure.

The methods and procedures for facial EMG data collection were the same as used in the experiment, described above.

Experimental Design.

The preliminary analysis used a 3 X 2 X 3 repeated measures factorial design, with one group factor (Anxious, Nonanxious, Control). The within groups repeated measures factors included Muscle (corrugator, zygomatic), and Affective Condition (neutral, happy, and sad faces). The

dependent variable was the mean of log-10 transformed RMS values of EMG activity, measured in microvolt units.

Data Analysis.

To assess whether the experimental procedures were sensitive to group differences in facial muscle pattern activity, an overall Group X Muscle X Affective Condition analysis of covariance (ANCOVA) was conducted (with mean baseline values for each affective condition as the covariates). An individual Group X Affective Condition ANCOVA was conducted for each muscle, to determine whether differences in patterns were specific to either muscle. Analyses of simple effects were conducted for each of the patient groups, to determine whether these groups showed the characteristic patterns of facial muscle activity evidenced by the control subjects.

A power analysis software program developed by John Nee, Ph.D., a statistician at New York State Psychiatric Institute ("EasyStat", unpublished), was employed to determine the minimum number of subjects necessary to detect a significant Group X Emotion interaction at either muscle site.

C. Results

Group Differences.

The results of the overall Group X Muscle X Affective Condition ANCOVA are shown in Figure 2. A highly significant Muscle X Affective Condition interaction, $F(2,75) = 16.19$, p

$< .00001$, indicated that the patterns of activity evoked by the affective faces differed for the corrugator and zygomatic muscles. There was also a nonsignificant trend for the groups to show different muscle activity patterns in response to the affective faces [Group X Muscle X Affective Condition interaction, $F(4,75) = 2.13$, $p = .08$]. Individual Group X Affective Condition ANCOVAs for the corrugator and zygomatic muscles were not significant.

Because of the trend for groups to show different muscle activity patterns (Figure 2), additional Group X Affective Condition ANCOVAs were conducted to further evaluate possible group differences. Analyses of simple effects revealed that there were no differences in the magnitude or pattern of EMG activity between the Nonanxious Depression and Normal Control groups, for either the corrugator, $F(2,55) = 1.09$, NS, or the zygomatic muscle, $F(2,55) = 0.17$, NS. This can be clearly seen in Figure 2. In contrast, the Anxious Depression group showed a different pattern of results. Unlike the Normal Control and Nonanxious Depression groups, the Anxious Depression group failed to show the characteristic differential patterns of activity in response to different affective faces [$F(2,19) = .81$, NS, for corrugator; $F(2,19) = .64$, NS, for zygomatic]. A significant difference in the pattern of corrugator muscle activity expressed by the Anxious and Nonanxious Depression groups, $F(2,41) = 3.43$, $p = .0419$, as well as a trend for zygomatic

pattern differences, $F(2,41) = 3.11$, $p = .0551$, indicated that the greatest muscle pattern differences are evident between the two patient subgroups.

Power Analysis.

Based on the outcome of the individual Group X Affective Condition ANCOVAs for the corrugator, $F(4,75) = 1.37$, $p = .2515$, and zygomatic muscles, $F(4,75) = 1.44$, $p = .2286$, the results of a power analysis revealed that a minimum of 52 subjects (52 total; i.e., about 17 per group) is necessary to detect a significant Group X Affect pattern interaction ($p < .05$) at either muscle site. This estimate was based on the assumption that a 1 standard deviation difference will be required to reveal meaningful differences between groups, with a power value of .80.

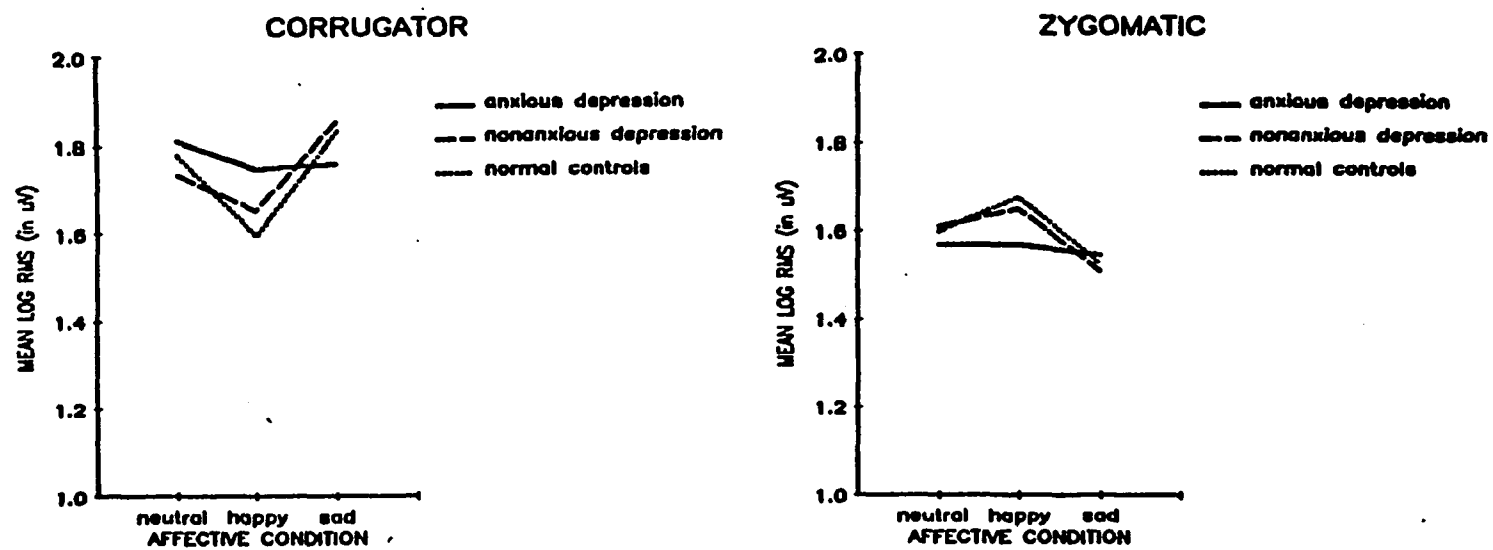
D. Discussion

The results of this preliminary analysis indicate that the EMG methods and data reduction procedures employed in the experiment are indeed sensitive to patient group differences. Even at this early stage, with relatively few patients per group, the significant differences and strong trends apparent between the Anxious and Nonanxious groups provide further rationale for distinguishing between these two depressive subtypes. The disordered pattern of facial muscle activity in the Anxious group, and the normal pattern of activity in the Nonanxious group, are consistent with the experimental hypotheses. Because of the similarity in

depressive symptom features between the two patient groups, the difference between groups appears to be due to the additional anxiety disorder (mainly panic anxiety) of the Anxious group. Although there was a trend toward handedness differences between groups, if this had affected muscle patterns, the bias would have been against the Nonanxious group (which contained 3 left-handers) rather than the Anxious group (which contained 2 left-handers).

The results of this preliminary analysis provide strong rationale for continuing with the proposed experimental design and methods. My proposal, therefore, is to increase group sizes to at least 17 subjects each, and to match groups for handedness in the completed experiment. The power analysis revealed that a total of 52 subjects is necessary to detect group differences at either muscle site, indicating that the proposed N of 17 subjects per group should be sufficient to detect group muscle pattern differences.

Figure 2. Group Comparison of Facial EMG Patterns.



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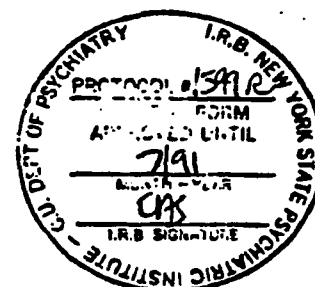
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Appendix D, 161-163

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Appendix E

Patient Consent Form - C



Purpose of Study

I understand that the purpose of this research is to learn more about hearing and other mental functions in patients with a condition like mine.

Procedures

Physiological activity from my hands, face and arms will be measured, while I am watching pictures on a screen. I will be asked to rate my feelings during the test session, and to fill out several forms which measure my feelings on other days. These tests will be given at Psychiatric Institute in a session of about 1 hour. I understand that as part of my participation in this project, I will be asked to retake the tests after about 6 weeks and possibly on one other occasion.

Risks & Benefits

This study is not designed for my benefit. I realize that it may lead to a greater understanding about what might cause and what might prevent conditions like mine. I have been informed that there are no anticipated physical or psychological risks involved in taking these tests.

Compensation

I will be paid at a rate of \$10 per hour for my participation in these tests.

Research Standards & Rights of Participants

I understand that participation in this project is voluntary and have been assured that I can refuse to participate or withdraw and this in no way affect my regular treatment or any other benefits to which I am entitled.

I understand that the test results will remain confidential. All data are kept in the laboratories of the Department of Biopsychology (New York State Psychiatric Institute) under lock and key, and only research staff and state/federal regulatory agency personnel have access to these data. In reporting the results of this study, privacy is protected by using coding systems that obscure the identity of individuals or by reporting only the average group results. This form will also be kept in a separate confidential file in the Department of Biopsychology.

If I have any questions about the study procedures, I may call Dr. Bruder at 960-2473. The New York State Psychiatric Institute - Columbia University Department of Psychiatry Institutional Review Board has approved the recruitment of subjects for this study. I understand that if I have any questions about my rights as a research subject or any complaints, I can call the Institutional Review Board at 960-5758. I have been given a copy of this consent form to keep.

Signature

Date

Informed Consent Addendum

Protocol Number 1599R

Patient Name: _____

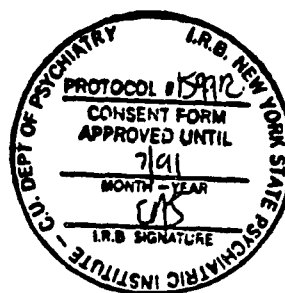
Principal Investigator: Dr. Gerard Bruder

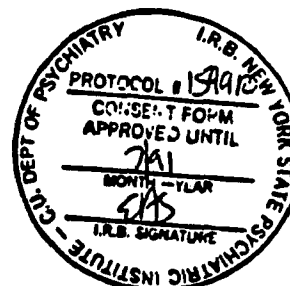
I have discussed the proposed research with this patient and, in my opinion, this patient understands the benefits and risks and is capable of freely consenting to participate in this research.

Signature _____

Date: _____

Member of Research Team





Non-Patient Consent Form - E

Purpose of Study

I understand that the purpose of this research is to learn more about physiological function.

Procedures

Physiological activity from my hands, face and arms will be measured, while I am watching pictures on a screen. I will be asked to rate my feelings during the test session, and to fill out several forms which measure my feelings on other days. These tests will be given at Psychiatric Institute in a session of about 1 hour. I understand that as part of my participation in this project, I will be asked to retake the tests after about 6 weeks and possibly on one other occasion.

Risks & Benefits

This study is not designed for my benefit. I have been informed that there are no anticipated physical or psychological risks involved in taking these tests.

Compensation

I will be paid at a rate of \$10 per hour for my participation in these tests.

Research Standards & Rights of Participants

I understand that participation in this project is voluntary and have been assured that I can refuse to participate or withdraw and this will in no way affect my regular treatment or any other benefits to which I am entitled.

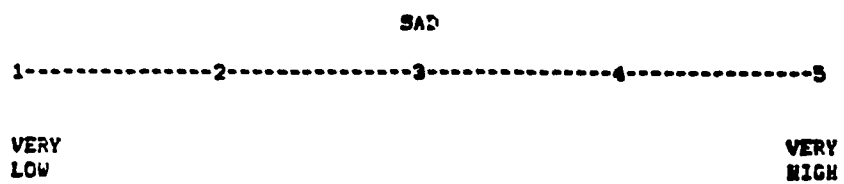
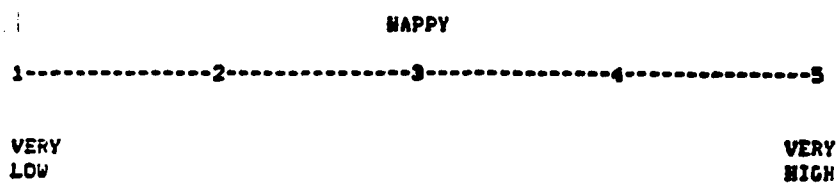
I understand that the test results will remain confidential. All data are kept in the laboratories of the Department of Biopsychology (New York State Psychiatric Institute) under lock and key, and only research staff and state/federal regulatory agency personnel have access to these data. In reporting the results of this study, privacy is protected by using coding systems that obscure the identity of individuals or by reporting only the average group results. This form will also be kept in a separate confidential file in the Department of Biopsychology.

If I have any questions about the study procedures, I may call Dr. Bruder at 960-2473. The New York State Psychiatric Institute - Columbia University Department of Psychiatry Institutional Review Board has approved the recruitment of subjects for this study. I understand that if I have any questions about my rights as a research subject or any complaints, I can call the Institutional Review Board at 960-5758. I have been given a copy of this consent form to keep.

Signature _____

Date _____

Appendix F



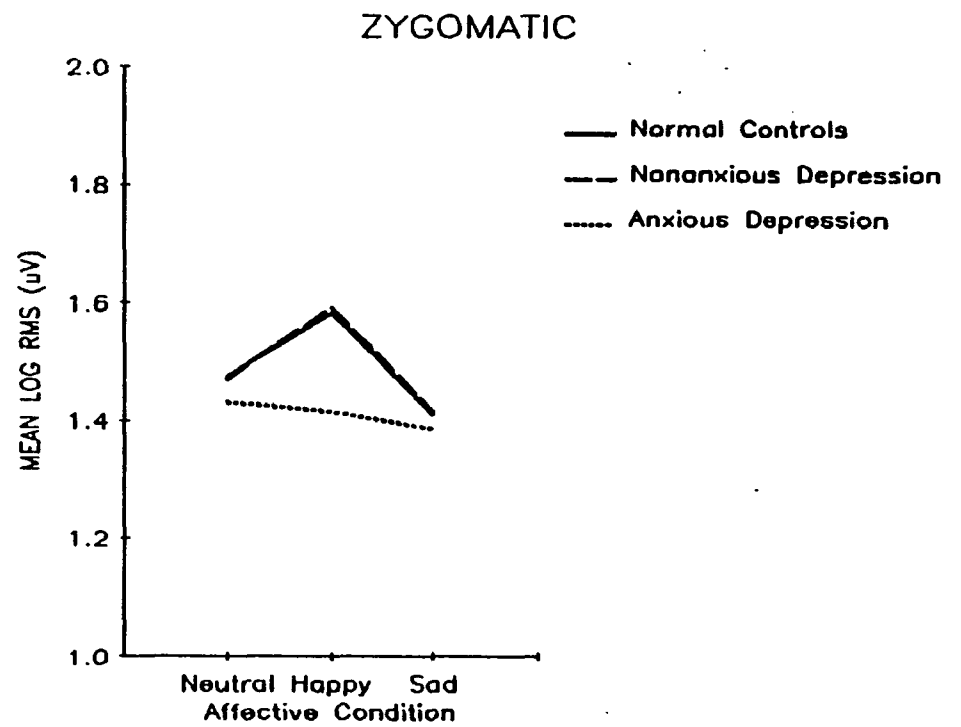
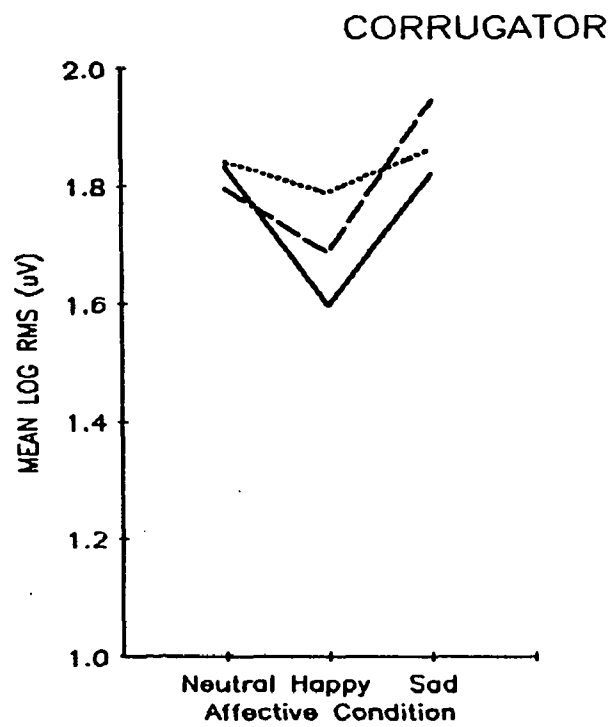
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Appendix L



References

- Ahern, G.L., & Schwartz, G.E. 1979. Differential lateralization for positive versus negative emotions. Neuropsychologia, 17, 693-698.
- Ahern, G.L., & Schwartz, G.E. 1985. Differential lateralization for positive and negative emotion in the human brain: EEG spectral analysis. Neuropsychologia, 23, 745-755.
- Akiskal, H.S. 1985. Anxiety: Definition, relationship to depression, and proposal for an integrative model. In A.H. Tuma & J. Maser (Eds.), Anxiety and the anxiety disorders (pp. 787-797). Hillsdale, NJ: Lawrence Erlbaum.
- Akiskal, H.S., Lemmi, H., Dickson, H., King, D., Yerevanian, B., & Van Valkenburg, C. 1984. Chronic depressions. Part 2. Sleep EEG differentiation of primary dysthymic disorders from anxious depressions. Journal of Affective Disorders, 6, 287-295.
- Akiskal, H.S., Lemmi, H., Yerevanian, B., King, D., & Belluomini, J. 1982. The utility of the REM latency test in psychiatric diagnosis: A study of 81 depressed outpatients. Psychiatry Research, 7, 101-110.
- American Psychiatric Association. 1987. Diagnostic and statistical manual of mental disorders, 3rd Edition, revised. Washington D.C.: American Psychiatric Association.
- Basmajian, J.V., & DeLuca, C.J. 1985. Muscles alive: Their functions as revealed by electromyography (5th ed.)

Baltimore: Williams & Wilkins.

Beck, A.T. 1963. Thinking and depression. Archives of General Psychiatry, 9, 324-333.

Beck, A.T. 1967. Depression: Clinical, experimental and theoretical aspects. New York: Harper & Row.

Beck, A.T. 1971. Cognition, affect and psychopathology. Archives of General Psychiatry, 24, 495-500.

Beck, A.T. 1978. The Beck Depression Inventory. Philadelphia: Center for Cognitive Therapy.

Benowitz, L.I., Bear, D.M., Mesulam, M.M., Rosenthal, R., Zaidel, E., & Sperry, R.W. 1983. Nonverbal sensitivity following lateralized cerebral injury. Cortex, 19, 5-12.

Berger-Gross, P., Bruder, G.E., Quitkin, F., & Goetz, R. 1985. Auditory laterality in depression: Relation to circadian patterns and EEG sleep. Biological Psychiatry, 20, 611-622.

Berger-Gross, P. & Bruder, G.E. 1984. Dichotic listening in the morning and evening. International Journal of Neuroscience, 22, 277-282.

Berlin, C.I., Hughes, L.F., Lowe-Bell, S.S., & Berlin, H.L. 1973. Dichotic right-ear advantage in children 5 to 13. Cortex, 9, 393-401.

Borod, J.C., & Caron, H.S. 1980. Facedness and emotion related to lateral dominance, sex, and expression type. Neuropsychologia, 18, 237-242.

Borod, J., & Koff, E. 1984. Asymmetries in affective

facial expression: Anatomy and behavior. In N. Fox & R. Davidson (Eds.), The psychobiology of affective development (pp. 293-323). Philadelphia, PA: Erlbaum & Associates.

Borod, J., Koff, E., & Buck, R. 1986. The neuropsychology of facial expression in normal and brain-damaged subjects. In P. Blanck, R. Buck, & R. Rosenthal (Eds.), Nonverbal communication in the clinical context. University Park, PA: Pennsylvania State University Press.

Borod, J.C., Koff, E., & Caron, H.S. 1983. Right hemispheric specialization for the expression and appreciation of emotion: A focus on the face. In E. Perecman (Ed.), Cognitive processes in the right hemisphere. New York: Academic Press.

Borod, J., Koff, E., Perlman, M., & Nicholas, M. 1983. Appreciation and expression of facial emotion in brain-damaged subjects. Paper presented at the International Neuropsychological Society Meeting, Mexico City, February.

Borod, J., Koff, E., Perlman-Lorch, M., & Nicholas, M. 1986. The expression and perception of facial emotion in brain damaged patients. Neuropsychologia, 24, 169-180.

Borod, J., Koff, E., & White, B. 1983. Facial asymmetry in posed and spontaneous expressions of emotion. Brain and Cognition, 2, 165-175.

Bowen, R.C., & Kohout, J. 1979. The relationship between agoraphobia and primary affective disorders. Canadian Journal of Psychiatry, 24, 317-322.

Breier, A., Charney, D.S., & Heninger, G.R. 1985. The diagnostic validity of anxiety disorders and their relationship to depressive illness. American Journal of Psychiatry, 142, 787-797.

Brown, S.L. 1981. Dissociation of pleasure in psychopathology. Journal of Nervous and Mental Disease, 169, 3-17.

Brown, S.L. 1982. The relationship between expressed and experienced emotion in depression. International Journal of Psychiatric Medicine, 12, 29-41.

Brown, S.L., & Schwartz, G.E. 1980. Relationships between facial electromyographic and subjective experience during affective imagery. Biological Psychology, 11, 49-62.

Brown, S.L., Schwartz, G.E., & Sweeney, D.R. 1978. Dissociation of self-reported and observed pleasure in depression. Psychosomatic Medicine, 40, 536-548.

Brown, S.L., Sweeney, D.R., & Schwartz, G.E. 1979. Differences between self-reported and observed pleasure in depression and schizophrenia. Journal of Nervous and Mental Disease, 167, 410-415.

Bruder, G.E. 1988. Dichotic listening in psychiatric patients. In K. Hugdahl (Ed.), Handbook of dichotic listening: Theory, methods and research (pp. 527-653). Chichester, England: Wiley.

Bruder, G.E., Quitkin, F.M., Stewart, J.W., Martin, C., Voglmaier, M.M., & Harrison, W.M. 1989. Cerebral laterality

and depression: Differences in perceptual asymmetry among diagnostic subtypes. Journal of Abnormal Psychology, 98, 177-186.

Bruder, G.E., Stewart, J.W., Voglmaier, M.M., Harrison, W.M., McGrath, P., Tricamo, E., & Quitkin, F.M. 1990. Cerebral laterality and depression: Relations of perceptual asymmetry to outcome of treatment with tricyclic antidepressants. Neuropsychopharmacology, 3(1), 1-10.

Bruder, G.E., Sutton, S., Berger-Gross, P., Quitkin, F., & Davies, S. 1981. Lateralized auditory processing in depression: Dichotic click detection. Psychiatry Research, 4, 253-266.

Bruder, G.E. & Voglmaier, M.M. 1990. Dichotic listening and subclinical depression. Paper presented at the International Neuropsychological Society Meeting, Orlando, Florida, February.

Bryden, M.D. 1982. Laterality: Functional asymmetry in the intact brain. New York: Academic Press.

Buck, R. 1980. Nonverbal behavior and the theory of emotion: The facial feedback hypothesis. Journal of Personality and Social Psychology, 38, 811-824.

Buck, R., & Duffy, R.J. 1980. Nonverbal communication of affect in brain-damaged patients. Cortex, 16, 351-362.

Cacioppo, J.T., & Petty, R.E. 1979. Attitudes and cognitive response: An electrophysiological approach. Journal of Personality and Social Psychology, 37, 2181-

2199.

Cacioppo, J.T., Petty, R.E., Losch, M.E., & Kim, H.S.
1986. Electromyographic activity over facial muscle regions can differentiate the valence and intensity of affective reactions. Journal of Personality and Social Psychology, 50, 260-268.

Campbell, R. 1978. Asymmetries in interpreting and expressing a posed facial expression. Cortex, 14, 327-342.

Carr, V., & Wale, J. The effects of antipsychotic drugs and symptomatology on perceptual asymmetry in schizophrenia. Journal of Neurology, Neurosurgery, and Psychiatry, 51, 1531-1537.

Carney, R.M., Hong, B.A., Brim, J., Plesons, D. & Clayton, P. 1983. Facial electromyography and reactivity in depression (brief communication). Journal of Nervous and Mental Disease, 171, 312-313.

Carney, R.M., Hong, B.A., Kulkarni, S., & Kapila, A.
1981a. A comparison of EMG and SCL in normal and depressed subjects. Pavlovian Journal of Biological Science, 16, 212-216.

Carney, R.M., Hong, B.A., O'Connell, M.F., & Amado, H.
1981b. Facial electromyography as a predictor of treatment outcome in depression. British Journal of Psychiatry, 138, 485-489.

Chapman, L.J., Chapman, J.P., & Raulin, M.L. 1976. Scales for physical and social anhedonia. Journal of Abnormal

Psychology, 85, 374-382.

Cicone, M., Wapner, W. and Garner, H. 1980. Sensitivity to emotional expressions and situations in organic patients. Cortex, 16, 145-158.

Clayton, P. 1987. Anxious depression: A reemerging subtype of depression. In G. Racagni & E. Smeraldi (Eds.) Anxious depression: Assessment and treatment. New York: Raven Press.

Cloninger, C.R., Martin, R.L., Clayton, P., & Guze, S.B. 1981. A blind follow-up and family study of anxiety neurosis: Preliminary analysis of the St. Louis 500. In D.F. Klein & J. Rabkin (Eds.) Anxiety: New research and changing concepts, pp. 137-154. New York: Raven Press.

Coffey, C.E. 1987. Cerebral laterality and emotion: The neurology of depression. Comprehensive Psychiatry, 28, 197-219.

Colbourn, C.J., & Lishman, W.A. 1979. Lateralization of function and psychotic illness: A left hemispheric deficit? In J. Gruzelier & P. Flor-Henry (Eds.), Hemisphere asymmetries of function and psychopathology (pp. 539-559). Amsterdam: Elsevier.

Cooley, E.L., & Nowicki, S. 1989. Discrimination of facial expressions of emotion by depressed subjects. Genetic, Social, and General Psychology Monographs, 115(4), 451-465.

Coyne, J.C. 1976a. Depression and the response of others.

Journal of Abnormal Psychology, 85, 186-193.

Coyne, J.C. 1976b. Toward an interactional description of depression. Psychiatry, 39, 28-40.

Curtis, G.C., Cameron, O.G., & Nesse, R.M. 1982. The dexamethasone suppression test in panic disorder and agoraphobia. American Journal of Psychiatry, 139, 1043-1046.

Damasio, A.R., Damasio, H., & Van Hoesen, G.W. 1982. Prosopagnosia: Anatomic basis and behavioral mechanisms. Neurology, 32, 331-341.

Darwin, C. 1872. The expression of emotion in man and animals. London: Murray.

Davidson, R. 1984. Hemispheric asymmetry and emotion. In K. Scherer & P. Ekman (Eds.), Approaches to emotion (pp. 39-57). Hillsdale, NJ: Erlbaum.

Davidson, R. 1985. Affect, cognition, and hemispheric specialization. In C.E. Izard, J. Kagan, & R. Zajonc (Eds.), Emotion, cognition, and behavior (pp. 320-365). New York: Cambridge University Press.

Davidson, R. 1987. Cerebral asymmetry and the nature of emotion: Implications for the study of individual differences and psychopathology. In R. Takahashi, P. Flor-Henry, J. Gruzelier, & S. Niwa (Eds.), Cerebral dynamics, laterality, and psychopathology (pp. 71-83). New York: Elsevier Science Publishers.

Davidson, R., & Tomarken, A.J. 1989. Laterality and

emotion: An electrophysiological approach. In F. Boller & J. Grafman (Eds.), Handbook of neuropsychology (Vol. 3, pp. 419-441). Amsterdam: Elsevier.

Davis, J.M. 1980. In H.I. Kaplan, A.M. Freedman, & B.J. Sadock (Eds) Comprehensive textbook of psychiatry, 3rd edition, Vol. 3., pp. 2290-2316. Baltimore: Williams & Wilkins.

DeJong, R.N. 1979. The neurological examination. Hagerstown, MD: Harper & Row.

DeKosky, S.T., Heilman, K.M., Bowers, D., & Valenstein, E. 1980. Recognition and discrimination of emotional faces and pictures. Brain and Language, 9, 206-214.

Dimberg, U. 1982. Facial reactions to facial expressions. Psychophysiology, 19, 643-647.

Dimberg, U. 1986. Facial reactions to fear-relevant and fear-irrelevant stimuli. Biological Psychology, 23, 153-161.

Dimberg, U. 1987. Facial reactions, autonomic activity and experienced emotion: A three-component model of emotional conditioning. Biological Psychology, 24, 105-122.

Dimberg, U. 1988. Facial electromyography and the experience of emotion. Journal of Psychophysiology, 2, 277-282.

Dimberg, U. 1989. Facial electromyography and facial emotional reactions. Uppsala Psychological Reports, 418. Sweden: Uppsala University.

Dimberg, U. 1990. Facial electromyography and emotional reactions. Psychophysiology, 27, 481-494.

Dimberg, U., & Lundquist, O. 1988. Facial reactions to facial expressions: Sex-differences. Psychophysiology, 25, 442-443.

Dimond, S.J., & Farrington, L. 1977. Emotional response to films shown to the right and left hemisphere of the brain measured by heart rate. Acta Psychologica, 41, 255-260.

Dopson, W., Beckwith, B., Tucker, D., & Bullard-Bates, C. 1984. Asymmetry of facial expression in spontaneous emotion. Cortex, 29, 243-251.

Drubach, D.A., & Kelly, M.P. 1989. Panic disorder associated with a right paralimbic lesion. Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 2, 282-289.

Ekman, P. 1975. Pictures of facial affect. Palo Alto: Consulting Psychologists Press, Inc.

Ekman, P. 1981. Methods for measuring facial action. In K. Scherer & P. Ekman (Eds.), Handbook on methods of nonverbal communications research. New York: Cambridge University Press.

Ekman, P., Hager, J. & Friesen, W.V. 1981. The symmetry of emotional and deliberate facial action. Psychophysiology, 18, 101-106.

Etcoff, N.L. 1984a. Perceptual and conceptual organization of facial emotions: Hemispheric differences. Brain and Cognition, 3, 385-412.

Etcoff, N.L. 1984b. Selective attention to facial identity and facial emotion. Neuropsychologia, 22, 281-295.

Etcoff, N.L. 1986. The neuropsychology of emotional expression. In: G. Goldstein and R. Tarter (Eds.) Advances in Clinical Neuropsychology. New York: Plenum Press.

Feinberg, T.E., Rifkin, A., Schaffer, C., & Walker, E. 1986. Facial discrimination and emotional recognition in schizophrenia and affective disorders. Archives of General Psychiatry, 43, 276-279.

Flor-Henry, P. 1976. Lateralized temporal-limbic dysfunction and psychopathology. Annals of the New York Academy of Sciences, 280, 777-795.

Flor-Henry, P. 1979. On certain aspects of the localization of the cerebral system regulating and determining emotions. Biological Psychiatry, 17, 677-698.

Fridlund, A.J. 1988. What can asymmetry and laterality in EMG tell us about the face and brain? International Journal of Neuroscience, 39, 53-69.

Fridlund, A.J., & Cacioppo, J.T. 1986. Guidelines for human electromyographic research. Psychophysiology, 23, 567-589.

Fridlund, A.J., Hatfield, M.E., Cottam, G.L., & Fowler, S.C. 1986. Anxiety and striate-muscle activation: Evidence from electromyographic pattern analysis. Journal of Abnormal Psychology, 95, 228-236.

Fridlund, A.J., & Izard, C.E. 1983. Electromyographic

studies of facial expression of emotions and patterns of emotion. In J.T. Cacioppo & R.E. Petty (Eds.), Social psychophysiology: A sourcebook (pp. 243-286). New York: Guilford Press.

Fried, I., Mateer, C., Ojemann, G., Wohns, R., & Fedio, P. 1982. Organization of visuospatial functions in human cortex. Brain, 105, 349-371.

Gaddum, J.H. 1945. Log-normal distributions. Nature, 156, 463-466.

Gainotti, G. 1972. Emotional behavior and hemispheric side of the lesion. Cortex, 8, 41-55.

Gatchel, R.J., McKinney, M.E., & Koebernick, L.F. 1977. Learned helplessness, depression, and physiological responding. Psychophysiology, 14, 25-31.

Ghardian, A.M., Gauthier, S., & Bertrand, S. 1986. Anxiety attacks in a patient with right temporal lobe meningioma. Journal of Clinical Psychiatry, 47, 270-271.

Goldstein, S.G., Filskov, S.B., Weaver, L.A., & Ives, J.O. 1977. Neuropsychological effects of electroconvulsive therapy. Journal of Clinical Psychology, 33, 798-806.

Golin, S., Hartman, S.A., Klatt, E.N., Munz, K., & Wolfgang, G.L. 1977. Effects of self-esteem manipulations on arousal and reactions to sad models in depressed and nondepressed college students. Journal of Abnormal Psychology, 86, 435-439.

Gray, J.A. 1982. The neuropsychology of anxiety: An

inquiry into the functions of the septo-hippocampal system.

Oxford: Oxford University Press.

Greden, J.F., Genero, N., & Price, H.L. 1985. Agitation-increased electromyogram activity in the corrugator muscle region: A possible explanation of the "Omega Sign"? American Journal of Psychiatry, 142, 348-351.

Greden, J.F., Genero, N., Price, H.L., Feinberg, M., & Levine, S. 1986. Facial electromyography in depression: Subgroup differences. Archives of General Psychiatry, 43, 269-274.

Greden, J.F., Price, H.L., Genero, N., Feinberg, M. & Levine, S. 1984. Facial EMG activity levels predict treatment outcome in depression. Psychiatry Research, 13, 345-352.

Halwes, T.G. 1969. Effects of dichotic fusion on the perception of speech. Supplement to Status Report on Speech Research. New Haven, CT: Haskins Laboratories.

Heller, W., & Levy, J. 1981. Perception and expression of emotion in right handers and left handers. Neuropsychologia, 19, 263-272.

Henriques, J.B., & Davidson, R.J. (in press). Left frontal hypoactivation in depression. Journal of Abnormal Psychology.

Henriques, J.B., & Davidson, R.J. 1990. Regional brain electrical asymmetries discriminate between previously depressed and healthy control subjects. Journal of Abnormal

Psychology, 99, 22-31.

Howes, M.J., & Hokanson, J.E. 1979. Conversational and social responses to depressive interpersonal behavior. Journal of Abnormal Psychology, 88, 625-634.

Izard, C.E. 1971. The face of emotion. New York: Appleton-Century-Crofts.

Izard, C.E. 1972. Patterns of emotions: A new analysis of anxiety and depression. New York: Academic Press.

Izard, C.E. 1977. Human emotions. New York: Plenum.

Izard, C.E. 1990. Facial expressions and regulation of emotions. Journal of Personality and Social Psychology, 58, 487-498.

Izard, C.E., & Malatesta, C.Z. 1987. Perspectives on emotional development I. Differential emotions theory of early emotional development. In J.D. Odofofsky (Ed.), Handbook of infant development (2nd ed., pp. 494-554). New York: Wiley Interscience.

Jaeger, J., Borod, J.C., & Peselow, E. 1986. Facial expression of positive and negative emotions in patients with unipolar depression. Journal of Affective Disorders, 11, 43-50.

Jaeger, J., Borod, J.C., & Peselow, E. 1987. Depressed patients have atypical hemispace biases in the perception of emotional chimeric faces. Journal of Abnormal Psychology, 96, 321-324.

Johnson, O., & Crockett, D. 1982. Changes in perceptual

asymmetries with clinical improvement in depression and schizophrenia. Journal of Abnormal Psychology, 91, 45-54.

Joseph, R. 1988. The right cerebral hemisphere. Journal of Clinical Psychology, 44, 631-673.

Kaplan, H.I., & Sadock, B.J. 1988. Synopsis of Psychiatry, 5th ed., p. 302. Baltimore: Williams & Wilkins.

Kent, J., Borod, J.C., Koff, E., Welkowitz, J., & Alpert, M. 1990. Posed facial emotional expression in brain-damaged patients. International Journal of Neuroscience, 43, 81-87.

Kimura, D. 1967. Functional asymmetry of the brain in dichotic listening. Cortex, 3, 163-178.

Klein, D.F. 1974. Endogenomorphic depression. Archives of General Psychiatry, 31, 447-454.

Kolb, B., & Milner, B. 1981. Observations on spontaneous facial expression after focal cerebral excisions and after intracarotid injection of sodium amytal. Neuropsychologia, 19, 505-514.

Kronfol, Z., deS. Hamsher, K., Digre, K., & Waziri, R. 1978. Depression and hemispheric functions: Changes associated with unilateral ECT. British Journal of Psychiatry, 132, 560-567.

Kuypers, H. 1958. Corticobulbar connections to the pons and lower brainstem in man. Brain, 81, 364-390.

Ladavas, E., Umiltà, C., & Ricci-Bitti, P.E. 1980. Evidence for sex differences in right-hemisphere dominance for emotions. Neuropsychologia, 18, 361-366.

Lader, M.H. 1975. Psychophysiology of clinical anxiety. In T. Silverstone & B. Barraclough (Eds.), Contemporary psychiatry. British Journal of Psychiatry, Special Publication, 9, 127-132.

Lader, M.H. 1980. Psychophysiological studies in anxiety. In G.D. Burrows & B. Davies (Eds.), Handbook of studies in anxiety. Amsterdam: Elsevier/North Holland.

Lang, P.J., Bradley, M.M., & Cuthbert, B.N. 1990. Emotion, attention, and the startle reflex. Psychological Review, 97, 377-395.

Leckman, J.F., Weissman, M.M., Merikangas, K.R., Pauls, D.L., & Prusoff, B.A. 1983. Panic disorder and major depression: Increased risk of depression, alcoholism, panic and phobic disorders in families of depressed probands with panic disorder. Archives of General Psychiatry, 40, 1055-1060.

Levin, S., Hall, J.A., Knight, R.A., & Alpert, M. 1985. Verbal and nonverbal expression of affect in speech of schizophrenic and depressed patients. Journal of Abnormal Psychology, 94, 487-497.

Levy, J., Heller, W., Banich, M.T., & Burton, L.A. 1983. Asymmetry of perception in free viewing of chimeric faces. Brain and Cognition, 2, 404-419.

Lewinsohn, P.M. 1974. Clinical and theoretical aspects of depression. In K.S. Calhoun, H.E. Adams, & K.M. Mitchell (Eds.) Innovative treatment methods in psychopathology. New

York: Wiley.

Lewinsohn, P.M. 1975. Engagement in pleasant activities and depression level. Journal of Abnormal Psychology, 84, 729-731.

Lewinsohn, P.M., & Libet, J. 1972. Pleasant events, activity schedules, and depression. Journal of Abnormal Psychology, 79, 291-295.

Lewinsohn, P.M., Lobitz, D., & Wilson, S. 1973. "Sensitivity" of depressed individuals to aversive stimuli. Journal of Abnormal Psychology, 81, 259-263.

Ley, R.G., & Bryden, M.P. 1979. Hemispheric difference in processing emotions and faces. Brain and Language, 7, 127-138.

Ley, R.G., & Bryden, M.P. 1982. A dissociation of right and left hemispheric effects for recognizing emotional tone and verbal content. Brain and Cognition, 1, 3-9.

Liebowitz, M.R., Qutikin, F.M., Stewart, J.W., McGrath, P.J., Harrison, W.M., Markowitz, J.S., Rabkin, J.G., Tricamo, E., Goetz, D.M. & Klein, D.F. 1988. Antidepressant specificity in atypical depression. Archives of General Psychiatry, 45, 129-137.

Liebowitz, M.R., Qutikin, F.M., Stewart, J.W., McGrath, P.J., Harrison, W.M., Rabkin, J., Tricamo, E., Markowitz, J.S. & Klein, D.F. 1984. Phenelzine vs. imipramine in atypical depression: A preliminary report. Archives of General Psychiatry, 120, 669-677.

Lucas, J.A., Bigler, E.D., & Telch, M.J. 1990. Memory dysfunction and panic disorder. Paper presented at the International Neuropsychological Society meeting, Orlando, Florida, February.

MacPhillamy, D.J., & Lewinsohn, P.M. 1974. Depression as a function of levels of desired and obtained pleasure. Journal of Abnormal Psychology, 83, 651-657.

Martin, C.C., Borod, J.C., Alpert, M., Brozgold, A., & Welkowitz, J. 1990. Spontaneous expression of facial emotion is schizophrenic and right-brain-damaged patients. Journal of Communication Disorders, 23, 287-301.

McKeever, W.F., & Dixon, M.F. 1981. Right-hemisphere superiority for discriminating memorized from nonmemorized faces: Affective imagery, sex, and perceived emotionality effects. Brain and Language, 12, 246-250.

Meadows, J.C. 1974. The anatomical basis of prosopagnosia. Journal of Neurology, 37, 489-501.

Moscovitch, M., & Olds, J. 1982. Asymmetries in spontaneous facial expressions and their possible relation to hemispheric specialization. Neuropsychologia, 20, 71-81.

Moscovitch, M., Strauss, E., & Olds, J. 1981. Handedness and dichotic listening performance in patients with unipolar endogenous depression who received ECT. American Journal of Psychiatry, 138, 988-990.

Nelson, C.A. 1987. The recognition of facial expressions in the first two years of life: Mechanisms of development.

Child Development, 58, 889-909.

Nelson, R.E., & Craighead, W.E. 1977. Selective recall of positive and negative feedback, self-control behaviors, and depression. Journal of Abnormal Psychology, 86, 379-388.

Oei, T.I., Verhoeven, W.M.A., Westenberg, H.G.M., Zwart, F.M., & van Ree, J.M. 1990. Anhedonia, suicide ideation, and dexamethasone nonsuppression in depressed patients. Journal of Psychiatric Research, 24, 25-35.

Oldfield, R.C. 1971. The assessment and analysis of handedness: The Edinburgh inventory. Neuropsychologia, 9, 97-113.

Ontiveros, A., Fontaine, R. Breton, G. Elie, R., Fontaine, S., & Dery, R. 1989. Correlation of severity of panic disorder and neuroanatomical changes of magnetic resonance imaging. Clinical and Research Reports, 1, 404-408.

Oscar-Berman, M., Zurif, E.B., & Blumstein, S.E. 1975. Effects of unilateral brain damage on the processing of speech sounds. Brain and Language, 2, 345-355.

Otto, M., Yeo, R.A., & Dougher, M.J. 1987. Right hemisphere involvement in depression: Toward a neuropsychological theory of negative affective experiences. Biological Psychiatry, 22, 1201-1215.

Pauls, D.L., & DiBenedetto, A.M. 1987. The familial relationship between panic disorder and major depressive disorder. In G. Racagni & E. Smeraldi (Eds.) Anxious

depression: Assessment and treatment. New York: Raven Press.

Paykel, E.S., Parker, R.R., Penrose, R.J.J., & Rassaby, E.R. Depressive classification and prediction of response to phenelzine. British Journal of Psychiatry, 134, 572-581.

Perrett, E.I., Rolls, E.T., & Caan, W. 1979. Temporal lobe cells of the monkey with visual responses selective for faces. Neuroscience Letters (Suppl. 3), S358.

Perrett, E.I., Rolls, E.T., & Caan, W. 1982. Visual neurones responsive to faces in the monkey temporal cortex. Experimental Brain Research, 47, 329-342.

Perris, C., Monakhov, K., Von Knorring, L., Botskarev, M., Nikiforov, A. 1978. A systemic structural analysis of the electroencephalogram of depressed patients: General principles and preliminary results of an international collaborative study. Neuropsychobiology, 4, 207-228.

Quitkin, F.M., Rabkin, J.G., Stewart, J.W., McGrath, P.J., Harrison, W., Davies, M., Goetz, R., & Puig-Antich, J. 1985. Sleep of atypical depressives. Journal of Affective Disorders, 8, 61-67.

Rapcsak, S.Z., Kaszniak, A.W., & Rubens, A.B. 1989. Anomia for facial expressions: Evidence for a category specific visual-verbal disconnection syndrome. Neuropsychologia, 8, 1031-1041.

Raskin, M., Peeke, H.V.S., Dickman, W., & Pinsker, H. 1982. Panic and generalized anxiety disorders: Developmental antecedents and precipitants. Archives of General

Psychiatry, 39, 687-689.

Rasmussen, T., & Milner, B. 1977. The role of early left-brain injury in determining lateralization of cerebral speech functions. In S.J. Dimond & D.A. Blizard (Eds.) Evolution and lateralization of the brain. Annals of the New York Academy of Sciences, 299, 355-369.

Reiman, E.M., Raichle, M.E., Butler, F.K., Herscovitch, P., & Robins, E. 1984. A focal brain abnormality in panic disorder, a severe form of anxiety. Nature, 310, 683-685.

Reiman, E.M., Raichle, M.E., Robins, E., Butler, F.K., Herscovitch, P., Fox, P., & Perlmutter, J. 1986. The application of positron emission tomography to the study of panic disorder. American Journal of Psychiatry, 143, 469-477.

Repp, B.H. 1977. Measuring laterality effects in dichotic listening. Journal of the Acoustical Society of America, 62, 720-737.

Reuter-Lorenz, P., & Davidson, R.J. 1981. Differential contribution of the two cerebral hemispheres to the perception of happy and sad faces. Neuropsychologia, 19, 609-613.

Rinn, W.E. 1984. The neuropsychology of facial expression: A review of the neurological and psychological mechanisms for producing facial expressions. Psychological Bulletin, 95, 52-77.

Robinson, R.G., Kubos, K.L., Starr, L.B., Rao, K., Price,

T.R. 1984. Mood disorders in stroke patients: Importance of location of lesion. Brain, 107, 81-93.

Robinson, R.G., & Starkstein, S.E. 1991. Heterogeneity in clinical presentation following stroke neuropathological correlates. Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 4, 4-11.

Rolls, E.T. 1981. Processing beyond the inferior temporal visual cortex related to feeding, memory, and striatal function. In Y. Katsuki, R. Norgren, & M. Sata (Eds.), Brain mechanisms of sensation (pp. 241-269). New York: Wiley.

Ross, E.D., & Mesulam, M.M. 1979. Dominant language functions of the right hemisphere? Prosody and emotional gesturing. Archives of Neurology, 36, 144-148.

Sacchetti, E., Conte, G., Calzeroni, A. 1987. The relationship between cerebral ventricular enlargement and anxiety symptomatology in major depression. In G. Racagni & E. Smeraldi (Eds.) Anxious depression: Assessment and treatment. New York: Raven Press.

Sacchetti, E., Conte, G., Calzeroni, A., Pennati, A., Battaglia, M., & Vita, A. 1987. The concept of anxious depression in relation to the biological heterogeneity of major affective disorder. In G. Racagni & E. Smeraldi (Eds.) Anxious depression: Assessment and treatment. New York: Raven Press.

Sackeim, H.A., Greenberg, M., Weiman, A., Gur, R., Hungerbuhler, J., & Geschwind, N. 1982. Functional brain

asymmetry in the expression of positive and negative emotions: Lateralization of insult in cases of uncontrollable emotional outbursts. Archives of Neurology, 19, 210-218.

Sackeim, H.A., & Gur, R.C. 1978. Lateral asymmetry in intensity of emotional expression. Neuropsychologia, 16, 473-481.

Sackeim, H.A., Gur, R.C., & Saucy, M.C. 1978. Emotions are expressed more intensely on the left side of the face. Science, 202, 434-436.

Schaffer, C.E., Davidson, R.J., & Saron, C. 1983. Frontal and parietal electroencephalogram asymmetry in depressed and nondepressed subjects. Biological Psychiatry, 18, 753-762.

Schaffer, C.E., Davidson, R.J., & Saron, C. 1984. Affective responses to faces: visual field asymmetries in depressed and non-depressed individuals. Paper presented at the International Neuropsychology Society Meeting, Houston, TX, February.

Schiff, B.B., & Lamon, M. 1989. Inducing emotion by unilateral contraction of facial muscles: A new look at hemispheric specialization and the experience of emotion. Neuropsychologia, 27, 923-935.

Schwartz, G.E., Ahern, G., & Brown, S. 1979. Lateralized facial muscle response to positive and negative emotional stimuli. Psychophysiology, 16, 561-571.

Schwartz, G.E., Brown, S., & Ahern, G. 1980. Facial

muscle patterning and subjective experience during affective imagery: Sex differences. Psychophysiology, 17, 75-82.

Schwartz, G.E., Fair, P.L., Salt, P., Mandel, M.R., & Klerman, G.L. 1976a. Facial expression and imagery in depression: an electromyographic study. Psychosomatic Medicine, 38, 337-347.

Schwartz, G.E., Fair, P.L., Salt, P., Mandel, M.R., & Klerman, G.L. 1976b. Facial muscle patterning to affective imagery in depressed and nondepressed subjects. Science, 192, 489-491.

Schwartz, G.E., Fair, P.L., Mandel, M.R., Salt, P., Mieske, M., & Klerman, G.L. 1978. Facial electromyography in the assessment of improvement in depression. Psychosomatic Medicine, 40, 355-360.

Schwartz, J., & Tallal, P. 1980. Rate of acoustic change may underlie hemispheric specialization for speech perception. Science, 207, 1380-1381.

Sheehan, D.V., Ballenger, J., & Jacobsen, G. 1980. Treatment of endogenous anxiety with phobic, hysterical, and hypochondriacal symptoms. Archives of General Psychiatry, 37, 51-59.

Sidtis, J.J. 1981. The complex tone test: Implications for the assessment of auditory laterality effects. Neuropsychologia, 19, 103-112.

Sidtis, J.J., & Volpe, B.T. 1988. Selective loss of complex-pitch or speech discrimination after unilateral

lesion. Brain and Language, 34, 235-245.

Sirota, A.D., & Schwartz, G.E. 1982. Facial muscle patterning and lateralization during elation and depression imagery. Journal of Abnormal Psychology, 91, 25-34.

Sirota, A.D., Schwartz, G.E., & Kirsteller, J.L. 1987. Facial muscle activity during induced mood states: Differential growth and carry-over of elated vs. depressed patterns. Psychophysiology, 24, 691-699.

Speaks, C., Niccum, N., & Carney, E. 1982. Statistical properties of responses to dichotic listening with CV nonsense syllables. Journal of the Acoustical Society of America, 72, 1185-1194.

Spielberger, C.D. 1983. The state-trait anxiety inventory. Palo Alto: Consulting Psychologists Press, Inc.

Spitzer, R.L., & Endicott, J. 1978. The schedule for affective disorders and schizophrenia: Lifetime version. New York State Psychiatric Institute.

Strack, S., & Coyne, J.C. 1983. Social confirmation of dysphoria: Shared and private reactions to depression. Journal of Personality and Social Psychology, 44, 798-806.

Strauss, E., & Moscovitch, M. 1981. Perception of facial expressions. Brain and Language, 13, 308-332.

Studdert-Kennedy, M., & Shankweiler, D. 1970. Hemispheric specialization for speech perception. Journal of the Acoustical Society of America, 48, 579-594.

Suarez, Y., Crowe, M.J., & Adams, H.E. 1978. Depression:

Avoidance learning and physiological correlates in clinical and analog populations. Behaviour Research and Therapy, 16, 21-31.

Suberi, M., & McKeever, W.F. 1977. Differential right hemispheric memory of emotional and non-emotional faces. Neuropsychologia, 15, 757-768.

Tassinari, L.G., Cacioppo, J.T., & Geen, T.R. 1989. A psychometric study of surface electrode placements for facial electromyographic recording: I. The brow and cheek muscle regions. Psychophysiology, 26, 1-16.

Taylor, C.B., & Arnow, B. 1988. The nature and treatment of anxiety disorders (pp. 137-182). New York: Free Press, Macmillan.

Tenke, C.E., Bruder, G.E., Sidtis, J.J., Towey, J., Erhan, H., & Voglmaier, M. 1989. Event related potential (ERP) asymmetries in a dichotic complex tone test (CTT). Society for Neurosciences Abstracts, Vol. 15, Part 1, p.477.

Tomkins, S.S. 1962. Affect, imagery, and consciousness. The positive affects. New York: Springer-Verlag.

Tucker, D.M. 1981. Lateral brain function, emotion, and conceptualization. Psychological Bulletin, 89, 19-46.

Tucker, D.M., Antes, J.R., Stenslie, C.E., & Barnhardt, T.N. 1978. Anxiety and lateral cerebral function. Journal of Abnormal Psychology, 87, 380-383.

Tucker, D.M., Stenslie, C.E., Roth, R.S., & Shearer, S.L. 1981. Right frontal lobe activation and right hemisphere

performance: Decrement during a depressed mood. Archives of General Psychiatry, 38, 169-174.

Van Valkenburg, C., Akiskal, H.S., Puzantian, V., & Rosenthal, T. 1984. Anxious depressions: Clinical, family history, and naturalistic outcome--comparisons with panic and major depressive disorders. Journal of Affective Disorders, 6, 67-82.

Vittone, B.J., & Uhde, T.W. 1985. Australia and New Zealand Journal of Psychiatry, 19, 330-341. Cited in: Pauls, D.L., & DiBenedetto, A.M. 1987. The familial relationship between panic disorder and major depressive disorder. In G. Racagni & E. Smeraldi (Eds.) Anxious depression: Assessment and treatment. New York: Raven Press.

Voglmaier, M.M. & Hakerem, G. 1989. Facial electromyography (EMG) in response to facial expressions: Relation to subjective emotional experience and trait affect. Paper presented at the Society for Psychophysiological Research meeting, New Orleans, October 18.

Wale, J. & Carr, V. 1988. Dichotic listening asymmetries and psychotic symptoms in schizophrenia: A preliminary report. Psychiatry Research, 25, 31-39.

Wale, J. & Carr, V. 1990. Differences in dichotic listening asymmetries in depression according to symptomatology. Journal of Affective Disorders, 18, 1-9.

Walker, E. 1981. Emotion recognition in disturbed and

normal children. Journal of Child Psychology and Psychiatry, 22, 263-268.

Walker, E., McGuire, M., & Bettles, B. 1984. Recognition and identification of facial stimuli by schizophrenics and patients with affective disorders. British Journal of Clinical Psychology, 23, 37-44.

Wall, M., Tuchman, M., & Mielke, D. 1985. Panic attacks and temporal lobe seizures associated with a right temporal lobe arteriovenous malformation: A case report. Journal of Clinical Psychiatry, 46, 143-145.

Watson, D., Clark, L.A., & Carey, G. 1988. Positive and negative affectivity and their relation to anxiety and depressive disorders. Journal of Abnormal Psychology, 97, 346-353.

Wexler, B.E. 1980. Cerebral laterality and psychiatry: A review of the literature. American Journal of Psychiatry, 137, 279-290.

Wexler, B.E. 1989. Perceptual and physiological responses to emotion-evoking stimuli in depression. Presented at the Samuel Sutton Psychophysiology and Psychopathology Colloquium, New York State Psychiatric Institute, December 12.

Wexler, B.E., & Heninger, G.R. 1979. Alterations in cerebral laterality during acute psychotic illness. Archives of General Psychiatry, 36, 278-284.

Yozawitz, A., Bruder, G., Sutton, S., Sharpe, L.,

Gurland, B., Gleiss, J., & Costa, L. 1979. Dichotic perception: Evidence for right hemisphere dysfunction in affective psychosis. British Journal of Psychiatry, 135, 224-237.

Zatorre, R.J. 1988. Pitch perception of complex tones and human temporal lobe function. Journal of the Acoustic Society of America, 84, 566-572.

Zuckerman, M. & Lubin, B. 1981. DACL: The Depression Adjective Check List. San Diego: Educational and Industrial Testing Service.

Zuckerman, M. & Lubin, B. 1985. MAACL-R: The Multiple Affect Adjective Check List-Revised. San Diego: Educational and Industrial Testing Service.

Zuckerman, M., Persky, H., & Curtis, G.C. 1968. Relationships among anxiety, depression, hostility, and autonomic variables. Journal of Nervous and Mental Disease, 146, 481-487.