Sympathetic reactivity to experimentally induced stress in alexithymia

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Abstract: Alexithymia refers to a specific disturbance in emotional processing that is manifested through difficulties in identifying and verbalizing feelings. The main objective of this research has been the study the relationship between sympathetic reactivity patterns, measured by electrodermal activity, and the alexithymia level, in a stressful laboratory situation. The subjects were high- or low-alexithymic young females (separated by TAS-20). The experiment involved six phases and the sympathetic reactivity was assessed in each one of them, using the Palmar Sweat Index (PSI), a sensitive indicator of the number of active sweat glands. The results show that both alexithymic and non-alexithymic subjects showed significant increases in PSI during stress phases and systematic decreases in all relaxation phases. We also obtained significant differences between TAS-20 low and high scores and between aggregated experimental phases, but no evidence of interaction TAS-20 x experimental phases. The results provide little support with the hypothesis that alexithymic subjects are more physiologically reactive to stress.

Key words: Emotion; alexithymia; stress; palmar sweat index.

Introduction

The term Alexithymia (literally from Greek: lack of words for emotions), refers to a specific disturbance in affective-emotional processing that is manifested through the following salient features: 1) difficulty in identifying and describing feelings and emotions verbally, 2) difficulty in distinguishing between feelings and somatic sensations that accompany emotional arousal, and 3) externally-oriented thinking and impaired symbolic activity (Taylor, 2000; Taylor, Bagby, & Parker, 1997). The most recent research has stressed the point that in alexithymia there is not only a difficulty in expressing emotions verbally but also a deficit in the cognitive processing of emotions (Berenbaum & Prince, 1994; Jessim & Markham, 1997; Lars-Gunnar & Simonsson-Sarnecki, 2002; Parker, Taylor, & Bagby, 1993; Roedema & Simons, 1999; Suslow, 1998; Suslow & Junghanns, 2002); as a consequence, emotions remain undifferentiated and poorly regulated (Taylor, Bagby, & Parker, 1991).

Experimental studies have supported the hypothesis that alexithymia might be associated with variations in brain activity (Franz, Schaefer, Schneider, Sitte, & Bachor, 2004; Richter et al. 2006). Kano et al. (2003) found differences in brain responses (mainly observed in the cortex areas, but not in the subcortical regions) to facial expressions between people with and without alexithymia. These results suggest that people with alexithymia process differently from people without alexithymia, and that this difference may account for the disorder of affect regulation and consequent peculiar behaviour in people with alexithymia.

These characteristics are conceptualized both as an affect-deficit disorder and a continuous personality trait (Martínez-Sánchez, Ato, Córcoles, Huedo, & Selva, 1998; Martínez-Sánchez, Ato, & Ortiz, 2003) that correlates positively with neuroticism (Pandey & Mandal, 1996), depression (Hendryx, Haviland, & Shaw, 1991) and anxiety (Bagby, Taylor, & Atkinson, 1988). Some authors (Horton, Gewirtz, & Kreutter, 1992) argue that alexithymia could be considered also as a state consequent to depression and/or anxiety (Hendryx, Haviland, Shaw, & Henry, 1994), as well as the effect of some chronic psychopathologic and somatic disorders.

It has been hypothesized that the limited emotional awareness and cognitive processing of affects lead to prolonged and amplifies the physiological arousal and neurovegetative reactivity to stress (Infrasca, 1997; Martin & Pohl, 1986b; Papeiak, Feurstein, & Spiegel, 1985) potentially disturbing the autonomic, pituitary-adrenal, and immune system. These dysregulated physiological responses over many years may result in untoward health effects (Li & Sinha, 2006; Lumley, Steptner, & Wehrm, 1996; Lumley, Tomakowsky, & Torosian, 1997).

Several studies report a higher prevalence of alexithymic characteristics among patients with stress-related disorders in comparison with other patients and normal controls (Fukunishi, Tsuruta, Hirabayashi, & Asukai, 2001; Kohn, Gurevich, Pickering, & McDonald, 1994; Krystal, Giller, & Cicchetti, 1986; Zeitlin, McNally, & Cassidy, 1993); however, the relationship between alexithymia and stress-related illness is more complex than a simple co-occurrence (Martin...
Several studies have examined autonomic activity associated with alexithymia, both at rest and in response to stressors. The hyperarousal model posits that alexithymia is related to higher tonic levels of sympathetic activity and/or exaggerated sympathetic reactivity (and possibly parasympathetic withdrawal) to emotional stressors. There is some evidence of tonic physiological hyperarousal in association with alexithymia (Gündel et al. 2002; Newton & Contrada, 1994; Papeciak, Feurestein, & Spiegel, 1985; Rabalías, 1987; Stone & Nielson, 2001). In contrast, the hypoarousal theory of alexithymia predicts that, under conditions of comparable emotional provocation, there is less physiological activation in individuals with alexithymic tendencies; there is no evidence that alexithymia leads to an excessive reactivity to stressors; indeed, most studies found either no alexithymia effect, or that alexithymia was related to less reactivity (Hyer, Woods, & Boudewyns, 1990; Linden, Lenz, & Stossel, 1996; Nemiah, Sifneos, & Apfel-Savitz, 1977; Wehmer, Brejnık, Lumley, & Stettner, 1995). In support of the hypoarousal model, four studies (i.e., Neumann, Sollers, Thayer, & Waldstein, 2004; Linden, Lenz, & Stossel, 1996; Newton and Contrada, 1994; Brejnık, Lumley, & Stettner, 1995) found that alexithymic men and women displayed attenuated heart rate responses to emotion-provoking tasks.

Because the studies using the SSPS and MMPI-based alexithymia scales are now considered to possess insufficient reliability and validity it was considered possible that the opposite results might be a function of different alexithymia measures.

Martin & Pihl (1986a) argued that alexithymic subjects are not necessarily more physiologically reactive to stress per se, but their subjective stress responses tend to be “decoupled” from their autonomic responses. Experimental evidence for a decoupling of HR and the subjective report of tension was found in alexithymics (Martin & Pihl, 1986a; Näring & van der Staak, 1995; Papeciak, Feurestein, & Spiegel, 1985). In a previous work (Martínez-Sánchez, Ortiz & Ato, 2001) we carried out a study to evaluate this “decoupling hypothesis”; results do not show significant correlations (high versus low alexithymia scores) between the subjective self-perception of activation and the autonomous reactivity.

The purpose of the present study was to evaluate the relationship between autonomic physiological reactivity patterns to stressful laboratory situations in college students with high versus low alexithymia scores. To produce emotional arousal in our subjects, we designed a laboratory stress task with four different experimental conditions: baseline (adaptation), relaxation and two conditions of stress, cognitive and visual stimuli.

Method

Subjects

Eighty-five female undergraduates psychology students, aged between 18 and 22 (\( \bar{x} = 19.38; s_x = 1.94 \)) participated in this experiment. They were all randomly selected from students enrolled in the “Psychology of Emotion” course at the University of Murcia who scored in the upper or lower quartile of the distribution of scores on the TAS-20. Subjects were divided into two groups according to their alexithymia scores which were based on quartile criteria, 20 in the lower 25 percentile, 23 in the upper 25 percentile. All subjects reported being in good health and none were taking medication at the time of the study that might have influenced either physiological responses or the perception of bodily sensations. Subjects were asked not to ingest alcohol, caffeine, or nicotine 2 hours prior to the experimental study. They all got an academic credit in return for their cooperation.

Materials and measures

The experiment was conducted in a laboratory with controlled temperature and illumination. Electrodermal activity was assessed using the Palmar Sweat Index (PSI); palmar sweat gland activity is a very sensitive indicator of autonomic reactivity (Fernández Castro et al., 1991; Freedman et al., 1994; Köhler, Weber, & Vögele, 1990; Torpin & Clements, 1993). PSI was assessed using the plastic impression technique, with fingerprints being obtained at 2.5-min intervals from the left index finger. PSI provides a direct measure of the number of sweat glands using a solution of 3% polyvinyl formal, 1% butylphthalate as a plasticizer, and the remainder ethylene dichloride.

Toronto Alexithymia Scale, TAS-20 (Bagby, Parker & Taylor, 1994), Spanish version (Martínez-Sánchez, 1996) was used. The TAS-20 is a 20-item self-report measure of the alexithymic construct with good internal consistency, high test-retest reliability and construct and criterion validity (Bagby, Taylor, & Parker, 1994; Taylor, Bagby, & Lumine, 2000). A three-factor structure is theoretically congruent with the alexithymia construct: (F.1) difficulty to identify feelings and to distinguish between feelings and somatic sensations of emotional arousal; (F. II) difficulty in describing feelings to others; and (F. III) externally-oriented thinking. A Spanish version of TAS-20 was accomplished by Spanish psychologists fluent in both English and Spanish, using back-translation methodology; this version showed good internal consistency (\( a = .78 \)) and test-retest reliability (\( r = .71, p< .001 \)) over a 19-week interval. These results are comparable to those obtained with the English version of the scale.
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The stability and replicability of this factor structure were demonstrated with both clinical and non-clinical populations by the use of confirmatory factor analysis (Bagby, Parker, & Taylor, 1994; Parker, Bagby, Taylor, Endler, & Schmitz, 1993).

Experimental tasks and procedure

The experiment protocol (i.e. baseline definition, data reduction recovery periods, etc.) follows the standard designs used in several studies (Fernández Castro, Martínez-Sánchez, & Ortiz, 1999; Köhler, Weber, & Vögele, 1990; Köhler & Troester, 1991; Köhler & Schusche, 1994; Martínez-Sánchez, Ortiz, & Fernández, 1998).

Participants attended individually the laboratory session. After orientation, the subject was greeted by the experimenter and given a written explanation of the experiment and consent form. After completion of the consent form, the subject sat quietly for 20 minutes. The experiment itself started with a 10 min adaptation phase during which the subjects had to relax and get used to the measurement procedures, especially the taking of the fingerprints (adaptation phase). This was followed by another 7.5 min of relaxation (from min 10 to 17.5), after which there was an instruction phase I lasting 1.5 min; from min 19 to 26.5 (stress I, mental arithmetic), the subjects has to count backward by step of seven as quickly as possible, starting from 2007, which was followed by another resting phase of 7.5 min duration (relaxation II from 26.5 to 34 min). The second part of the experiment followed the same scheme: 34-35 instruction phase II; 35-42.5 (stress II) during this phase the subjects had to watch a distressing film about surgery. Thereafter the subjects were told to relax again (III-relaxation from 42.5-50 min). All except 5 students completed the experimental protocol.

Results

Due to the high heterogeneity between groups variance observed in almost all phases of the experiment, we carried out Bartlett’s homogeneity of variance tests for scores obtained in each phase (see Table 1). A transformation log of observed electrodermal activity (PSI scores) was applied in order to get homogeneous groups.

Table 1: Bartlett’s homogeneity of variances tests of all phases, before and after log transformation.

<table>
<thead>
<tr>
<th>Phase</th>
<th>Before transformation</th>
<th>After transformation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bartlett $\chi^2$</td>
<td>$p$</td>
</tr>
<tr>
<td>Adaptation</td>
<td>2.99</td>
<td>.084</td>
</tr>
<tr>
<td>Relaxation 1</td>
<td>8.45</td>
<td>.004</td>
</tr>
<tr>
<td>Stress 1</td>
<td>6.85</td>
<td>.009</td>
</tr>
<tr>
<td>Relaxation 2</td>
<td>0.10</td>
<td>.752</td>
</tr>
<tr>
<td>Stress 2</td>
<td>8.04</td>
<td>.005</td>
</tr>
<tr>
<td>Relaxation 3</td>
<td>1.16</td>
<td>.281</td>
</tr>
</tbody>
</table>

To cope with the resulting 2 (TAS classes: Low and High Alexithymia) x 3 (Phases: Adaptation, Relaxation and Stress) mixed design with repeated measures in the second variable, we used a mixed model approach (Keselman, Algina, & Kowalchuk, 2001). This analytical strategy unifies many common statistical approaches, including standard repeated measures, in a general likelihood-based procedure called general linear mixed models (Pinheiro & Baltes, 2000; Verbeke & Molenberghs, 1997). It basically consists of a conventional linear model to test repeated measures hypotheses combined with a flexible specification of the most appropriate covariance structure. Modelling the correct covariance structure of the data should normally result in more powerful tests of fixed-effect parameters (Littell, Pendergast, & Natarajan, 2000).

Mean values of all experimental phases with original PSI measure show a clear trend to a systematic increase of the dependent measure in all stress phases, and systematic decreasing in all relaxation phases, but no evidence of interaction in function of TAS scores (see Table 2 and Figure 1). The same pattern was observed when all stress and relaxation phases were aggregated (see Table 3 and Figure 2).

Table 2: Observed means of all experimental phases by TAS scores; Experiment phases

<table>
<thead>
<tr>
<th>TAS score</th>
<th>Adaptation</th>
<th>1-S</th>
<th>2-S</th>
<th>3-S</th>
<th>Relax.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>4.635</td>
<td>2.987</td>
<td>7.890</td>
<td>2.912</td>
<td>6.634</td>
</tr>
</tbody>
</table>

Figure 1. Means of all experimental phases

Table 3: Observed means of aggregated experimental phases.

<table>
<thead>
<tr>
<th>TAS score</th>
<th>Adaptation</th>
<th>Relaxation</th>
<th>Stress</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>4.635</td>
<td>2.913</td>
<td>7.262</td>
</tr>
<tr>
<td>High</td>
<td>6.646</td>
<td>3.945</td>
<td>11.338</td>
</tr>
</tbody>
</table>

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We used PROC MIXED procedure in SAS version 8 (SAS Institute, 1996) to estimate fixed effect parameters. In order to compare models with the same fixed effects we tried different covariance structures with our data. The best covariance structure, in terms of both indexes of relative goodness-of-fit Akaike Information (AIC) and Schwartz's Bayesian Criterion (SBC), was compound symmetry (see Table 4). Models with closer to zero AIC and SBC criteria indicate a better fit.

Table 4: Values of Akaike and Schwarz criteria on 7 covariance structures

<table>
<thead>
<tr>
<th>Covariance structure</th>
<th>Akaike’s criterion</th>
<th>Schwarz’s criterion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compound symmetry</td>
<td>-229.1</td>
<td>-230.8</td>
</tr>
<tr>
<td>Toeplitz</td>
<td>-229.2</td>
<td>-234.2</td>
</tr>
<tr>
<td>Huynh-Feldt</td>
<td>-229.9</td>
<td>-235.6</td>
</tr>
<tr>
<td>Homogeneous</td>
<td></td>
<td></td>
</tr>
<tr>
<td>compound symmetry</td>
<td>-231.9</td>
<td>-237.6</td>
</tr>
<tr>
<td>Unstructured</td>
<td>-232.0</td>
<td>-249.2</td>
</tr>
<tr>
<td>Autoregressive (1)</td>
<td>-240.7</td>
<td>-242.3</td>
</tr>
<tr>
<td>Variance components</td>
<td>-259.8</td>
<td>-260.6</td>
</tr>
</tbody>
</table>

Using type 3 Sum of Squares for fixed effects, we obtained significant differences between TAS Low and High scores: $F_{1,36} = 4.63; p = .0382$ and between aggregated experimental phases: $F_{2,74} = 79.54; p < .0001$, but no evidence of interaction TAS x experimental phases.

**Discussion**

The main objective of this study has been to evaluate the autonomic reactivity to experimentally induced stress in alexithymic subjects. The results show that both alexithymic and nonalexithymic subjects showed significant increases in PSI during the stress phases; mean values of all experimental phases with original PSI measure show a clear trend to a systematic increase of dependent measure in all stress phases, and systematic decreasing in all relaxation phases, but no evidence of interaction in function of TAS-20 scores; the same pattern was observed when all stress and relaxation phases were aggregated. Also, we obtained significant differences between TAS-20 low and high scores and between aggregated experimental phases, but no evidence of interaction TAS-20 x experimental phases.

High alexithymics showed an increase of electrodermal activity as an expression of autonomous arousal during stress phase. These results provide little support to the hypothesis that alexithymic subjects are more physiologically reactive to stress, evaluated with the Palmar Sweat Index.

The association between alexithymia and higher sympathetic vegetative reactivity (emotional arousal) is still controversial. Our data confirm Papciak et al.’s (1985) findings. They predicted that alexithymic male college students would react autonomically to stress just as normal subjects do; they used a stress quiz to provoke autonomic responses and recorded blood pressure, HR, and frontal electromyogram results, founding that both alexithymic and nonalexithymic subjects showed significant increases in HR and blood pressure during the stress quiz. Stone & Nielson (2001) found that high-alexithymic subjects had a significantly higher electrodermal activity after exposure to an emotionally arousing videotape than that of the low-alexithymic participants, but no differences were found between high and low alexithymics in electrodermal activity under cognitive distress.

With the exception of Waldstein et al. (2002), six studies supporting the hypoarousal model (Linden et al., 1996; Neumann et al., 2004; Nemiah et al., 1997; Newton and Contra, 1994; Wehmer et al., 1995) consisted of considerably younger participants which may, at least in part, account for the differential reactivity patterns (Neumann et al., 2004). In an investigation of alexithymia and cardiovascular reactivity Linden et al. (1996) found no relationship between alexithymia and blood pressure responses, although heart rate responses were lower in high alexithymia individuals. In a similar study, Friedlander et al. (1997) found no relationship between alexithymia and blood pressure responses to the stressor of viewing disgusting scenes, but women with high alexithymia had a different pattern of heart rate changes than women with low alexithymia.

Only a few studies have provided evidence of sympathetic hyperactivity in alexithymics in response to emotional stimuli. A possible explanation for these different results may be that different types of alexithymia, formed by distinct neural malfunction produce different levels of sympathetic activity (Larsen, Brand, Bermond, & Hijman, 2003). The inconsistent findings may be explained, in part, by the use of different emotion-inducing stimuli and also to the monitoring of different physiological variables.

Regarding changes of PSI during stressful situations, our results replicate findings from previous studies (Köhler, Weber, & Vögele, 1990; Köhler & Troester, 1991; Köhler & Schusshel, 1994; Martínez-Sánchez, Fernández, & Ortiz, 1998; Turpin & Clements, 1993), in accordance with reac-
tions in other psychophysiological variables observed in some studies: PSI increased significantly from baseline to stress and decreased after its cessation.

There are some limitations in the present study concerning the generalization of results. First, the subjects were women and the applicability of the results for the male population remains to be shown. Second, there may be some limitations in the generalization of the results related with PSI; further studies are needed to replicate changes in PSI during stress, however diverse investigations have demonstrated that PSI displays essentially the same behaviour as electrodermal variables (Clements & Turpin, 1996); the physiologic basis of the electrodermal response has been identified as the activity of the sudoriferous glands, which are innervated by the sympathetic branch, thus making this neurovegetative parameter reliable for measuring arousal. Another problem is the intrusiveness of method, because the subject perceives clearly the application of the drop, its removal and the social interactions involved.

In conclusion, it must be concluded that our data provides little support with the hypothesis that alexithymia are more physiologically reactive to experimentally induced stress.

References


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