

Alexithymia, somatosensory amplification and counter-dependency in patients with chronic pain

ISMAIL AK *, KEMAL SAYAR and TULIN YONTEM

Department of Psychiatry, Karadeniz Technical University School of Medicine, Farabi Hospital, 61080 Trabzon, Turkey

summary

There is a vast body of research concerning the psychological characteristics of patients having chronic benign pain. Recently, the constructs of alexithymia, somatosensory amplification and counter-dependency have been studied in chronic pain patients. These studies have provided discrepant findings. In the present study, the authors investigate whether chronic pain patients differ from healthy controls in terms of these relatively new constructs. We aimed to see whether any of these constructs may be a 'psychological marker' to differentiate chronic pain group from healthy people. In the study, 30 chronic pain patients were compared to 30 healthy controls. All the patients had suffered from daily back, extremity or head aches for at least the previous six months. Patients were assessed with the Toronto Alexithymia Scale, the Somatosensory Amplification Scale and the Counter-dependency Scale as well as with a detailed sociodemographic questionnaire. Chronic pain patients were found to amplify their somatic sensations more than healthy controls, and they also had more difficulty in identifying their feelings and distinguishing them from bodily sensations of emotion. Chronic pain patients with a history of psychiatric disorders also amplified their somatic sensations and at the same time had more difficulty in identifying their feelings than patients with no history of psychiatric disorders. Measures of counter-dependency were not able to distinguish between chronic pain patients and healthy controls. In conclusion, chronic pain patients have difficulty in identifying feelings which in turn makes them prone to somatic expressions of psychic distress. Those individuals who introspect and who have a tendency to select and focus on relatively weak or infrequent sensations (the main elements of somatosensory amplification) might be more prone to chronic pain.

Key words: Chronic pain; alexithymia; somatosensory amplification; counter-dependency.

introduction

There is a vast body of research concerning the psychological characteristics of patients having chronic benign pain. Most research is focused on the relationship of depression and chronic pain, investigating a possible causal relationship between these two constructs. Recently, the concept of somatosensory amplification has been applied to the chronic pain population to explain how maladaptive cognitions may lead to heightened pain perception.¹ Somatosensory amplification refers to a tendency to experience somatic and visceral sensations as usually intense, noxious and disturbing. It involves bodily hypervigilance, the predisposition to focus on certain weak and infrequent bodily sensations, and a tendency to appraise them as pathological and symptomatic of disease, rather than normal.^{2,3} Some studies indicate that amplification of benign bodily sensations may be related to the more general process of somatization rather than being restricted to hypochondriasis.⁴ In

*To whom correspondence should be addressed. E-mail: iak@meds.ktu.edu.tr and iakktu@yahoo.com

chronic pain populations, somatosensory amplification may be more determined by levels of anxiety and depression than by pain *per se*.⁵ According to Gregory *et al.* subjects with chronic pain that included locations like head, chest, abdomen or pelvis had significantly higher scores of somatosensory amplification scale (SSAS) than subjects having chronic pain only in their back and/or extremities.⁶ Another construct that is investigated in chronic pain samples is alexithymia. Alexithymia is a personality construct characterized by difficulty in identifying and communicating feelings, and externally oriented thinking.⁷ Alexithymic individuals may tend to misinterpret their emotional arousal as symptoms of physical illness.⁸ Unable to identify affects as signals of inner psychic events, alexithymic individuals are thought to focus on, and to amplify, the somatic sensations of emotional arousal, which are then experienced as overwhelming somatic distress and/or misinterpreted as signs of disease.⁹ A study of psychiatric outpatients found that reporting of somatic symptoms was strongly correlated with alexithymia.¹⁰ Initial studies in chronic pain patients reported increased prevalence of alexithymia.¹¹ Several recent studies of chronic pain have used the construct-derived and well-validated Toronto Alexithymia Scale (TAS) or its recent 20-item version. In a sample of motor vehicle accident survivors with chronic pain¹² and in a sample of heterogeneous chronic pain patients,¹³ alexithymia appeared to be increased but, as Lumley *et al.*¹⁴ suggest, the lack of comparison groups in these studies limits conclusions about the relationship between alexithymia and chronic pain. In a recent study, chronic pain patients had significantly lower scores of alexithymia compared to psychiatric controls.⁵ Chronic non-malignant pain patients were compared to healthy controls in another study and no significant difference in terms of alexithymia was found between these two groups.⁶ In a similar study conducted in Turkey, no significant difference was found between chronic pain patients and healthy controls in terms of alexithymia.¹⁵ To demonstrate that alexithymia is associated with chronic pain rather than with the status of being a patient, pain patients were compared with patients who seek treatment for nicotine dependence and obesity.¹⁴ Chronic pain patients were more alexithymic than nicotine-dependent or obese patients; the latter two groups did not differ. This study demonstrated that alexithymia is increased among patients with chronic pain and this relationship is not confounded by a treatment-seeking bias.¹⁴ As may be seen from the reviewed literature, there is still some controversy on the relationship between alexithymia and chronic pain. This controversy may be attributed to the impact of psychiatric comorbidity on the measure of alexithymia. A more recent construct studied in the chronic pain population has been the construct of counter-dependency. First coined by Barsky¹⁶ to summarize personality characteristics of patients with chronic pain, the construct was then developed further and was measured quantitatively by Likert-type scale by Gregory and Berry.¹⁷ Counterdependent patients are characterized by emotional suppression, idealization of relationships, strong work ethic, caregiving role identity, and self-reliance.^{6,17} Authors have validated a scale to describe these characteristics (counter-dependency scale) and counter-dependency traits were found to be strongly associated with chronic pain and appeared independent of anxiety, depression, or psychiatric morbidity.¹⁷

In the present study, we investigated whether chronic pain patients differ from healthy controls in terms of these new constructs. By employing measures of somatosensory amplification, alexithymia and counterdependency we want to see whether any of these constructs may be a 'psychological marker' to differentiate chronic pain group from healthy people. We also

investigate the possible impact of past psychiatric disorders on any of these measures.

method

Subjects

Thirty consecutive chronic pain patients who sought help from the Karadeniz Technical University Medical School Hospital outpatient clinics formed the study group. Karadeniz Technical University Hospital in Trabzon city is located in North Eastern Turkey and serves some five million people from the surrounding area. All consecutive patients who were referred from other specialty outpatient clinics to the chronic pain outpatient clinics between 1 January 2002 and 31 March 2002 were interviewed for the study. Illiterate subjects unable to comprehend and fill in the questionnaires were excluded. Also excluded were patients whose cause of pain was a malignancy, patients with a primary psychotic disorder or a cognitive impairment. All the patients who formed the study group had suffered from daily back, extremity or head aches for at least the previous six months. Thirty healthy controls who had not reported any medical disorder or pain during the previous six months were also recruited. Both groups comprised 25 females and 5 males, the mean \pm standard deviation ages for the pain and healthy control groups being 40.6 ± 11.4 and 40.2 ± 14.4 ($t = 0.12$, $p > 0.05$), respectively. There was no significant difference between the educational ($\chi^2 = 4.8$, $p > 0.05$) or marital status ($\chi^2 = 0.11$, $p > 0.05$) of the subjects. Within the study group, the mean duration of pain was 8.1 ± 7.1 months. Six patients suffered from headaches, 14 from back pain and 10 from pain in the extremities. The study group comprised 21 housewives, 6 workers and 3 private business holders whereas there were 16 housewives, 12 workers and 2 private business holders in the healthy control group. Socioeconomic status of the patients as reported by themselves was as follows: in the study group, 4 poor, 24 moderate, 2 good and the control group 9 poor, 17 moderate and 4 good. The difference was not statistically significant ($\chi^2 = 3.7$, $p > 0.05$).

Measures

Patients provided informed consent and completed the following measures during the initial evaluation apart from a detailed sociodemographic form completed by the physician during the interview.

A 10 cm visual analog scale (VAS), the extremities of which represented 'no pain' and 'worst possible pain', was used by the patients to rate their pain intensity. Most studies that compare VAS with numerical and verbal ratings conclude that the VAS or the numerical ratings are statistically preferable to the verbal rating scales.¹⁸

The Toronto Alexithymia Scale-20 items (TAS) is a 20-item self-report scale which has good psychometric properties.^{19,20} Subjects are asked to judge on a 5-point Likert scale the extent to which they agree or disagree with each statement. The results are expressed as TAS-20 global scores, as well as three subscales measuring difficulty in identifying feelings and distinguishing them from bodily sensations of emotion (Factor 1), difficulty expressing feelings (Factor 2), and externally oriented thinking (Factor 3). The Turkish translation of the TAS-20 has good reliability ($\alpha = 0.76$)²¹. Items are rated on a 1–5 scale and summed; higher scores indicate greater levels of alexithymia.

The Somatosensory Amplification Scale (SSAS) is a 10-item self-report questionnaire that evaluates the tendency to experience ordinary bodily and visceral sensation as intense, noxious and disturbing.²² The respondent rates the degree to which each statement is 'characteristic of you in general',

on an ordinal scale from 1 to 5, labeled from 'not at all' to 'extremely'. Higher scores on the SSAS were found in hypochondriacal patients as well as in patients making frequent use of medical care.⁴ We prepared a Turkish translation of the SSAS, which was checked by blind back-translation and very good internal reliability with Cronbach's $\alpha = 0.80$.

The Counterdependency Scale (CDS) is a 5-item scale developed by Gregory and Berry¹⁷ after clinical observations that a large subgroup of chronic pain patients did not follow patterns frequently cited in the literature of dependency, neediness, and depression.⁶ These patients appeared to minimize emotional distress; describe idealized, shallow relationships with stereotyped roles; and lead overly productive lives until the development of their pain syndrome. We used a Turkish translation of the CDS, which was checked by blind back-translation and moderate internal reliability with Cronbach's $\alpha = 0.60$. In our study, the sum of all items was considered the measure of CDS.

Statistics

Groups were compared at the bivariate level using *t*-tests for continuous variables and chi-squared tests for categorical variables. Chronic pain patients were dichotomized according to the presence of past psychiatric disorder and compared in between with chi squared and Mann-Whitney *U*-tests.

results

There was no statistically significant difference between the age, gender, socioeconomic and marital status of the two groups (statistics given in the Subjects section). While sixteen patients (53%) reported a history of psychiatric disorders there were only 3 subjects from the control group (10%). The difference was statistically significant ($\chi^2 = 13.0$, $p < 0.001$). Six patients (20%) and 3 controls (10%) reported a family history of psychiatric disorder, but the difference was not significant ($\chi^2 = 1.1$, $p > 0.05$).

Chronic pain patients scored significantly higher than healthy controls on the TAS-20 (particularly on the difficulty in identifying feelings subscale) and the SSAS. The only subscale of TAS-20 which contributed to the difference was the difficulty in identifying feelings and distinguishing them from bodily sensations of emotion subscale. There was no statistically significant difference in the subscales of inability to express feelings or external oriented thinking between the two groups. Also non-significant was the difference in the CDS (Table I).

Chronic pain patients were dichotomized as those having a psychiatric disorder history or not and compared in between. Sixteen patients with a history of psychiatric disorders were compared to 14 patients with no history of psychiatric disorders. Those with a psychiatric disorder history scored significantly higher on the measures of TAS total, TAS-1 (difficulty identifying feelings) and SSAS. There was no significant difference in CDS (Table II).

Patients with a history of psychiatric disorder did not differ significantly from those without in terms of pain localization, pain severity, socioeconomic status and duration of pain ($p > 0.05$). The mean age of the first group was significantly higher than the latter (45.1 ± 6.4 versus 38.0 ± 9.7 , $z = 2.27$, $p = 0.02$).

In summary, chronic pain patients were found to amplify their somatic sensations more than healthy non-patient controls and they had more difficulty in identifying their feelings and distinguishing them from bodily sensations of

Table I.
Comparison of psychological variables between chronic pain patients and healthy controls

	Mean ± SD Patient (n = 30)	Mean ± SD Controls (n = 30)	t	p
SSAS	35.5 ± 7.4	29.3 ± 6.6	3.37	0.001
TAS-1	20.1 ± 6.7	14.9 ± 5.9	3.154	0.003
TAS-2	13.8 ± 5.0	12.4 ± 3.6	1.206	0.233
TAS-3	21.0 ± 5.0	21.1 ± 3.7	0.146	0.885
TAS-Total	54.9 ± 11.3	48.5 ± 10.3	2.298	0.025
CDS	11.1 ± 3.3	10.5 ± 3.6	0.589	0.558

SSAS: Somatosensory Amplification Scale, TAS 1: the Toronto Alexithymia Scale, ‘difficulty identifying feelings’ subscale, TAS 2: the Toronto Alexithymia Scale ‘difficulty in expressing feelings’ subscale, TAS 3: the Toronto Alexithymia Scale, ‘externally oriented thinking’ subscale, TAS-Total: the Toronto Alexithymia Scale total score, CDS: the Counter-dependency Scale.

Table II.
Comparison of psychological variables between chronic pain patients with a prior psychiatric history and chronic pain patients without a past psychiatric history

	Patient with psychiatric history (n = 16) Mean ± SD	Patient without psychiatric history (n = 14) Mean ± SD	z	p
SSAS	39.50 ± 6.36	31.00 ± 6.01	3.129	0.002
TAS-1	22.68 ± 6.46	17.28 ± 6.13	2.165	0.030
TAS-2	15.18 ± 4.94	12.21 ± 4.75	1.733	0.083
TAS-3	21.18 ± 4.62	20.78 ± 5.64	0.000	1.000
TAS-total	59.06 ± 12.53	50.28 ± 7.79	2.143	0.032
CDS	11.43 ± 3.03	10.71 ± 3.68	−0.752	0.452

SSAS: Somatosensory Amplification Scale, TAS 1: the Toronto Alexithymia Scale, ‘difficulty identifying feelings’ subscale, TAS 2: the Toronto Alexithymia Scale ‘difficulty in expressing feelings’ subscale, TAS 3: the Toronto Alexithymia Scale, ‘externally oriented thinking’ subscale, TAS-Total: the Toronto Alexithymia Scale total score, CDS: the Counter-dependency Scale.

emotion. Chronic pain patients with a history of psychiatric disorder also amplified their somatic sensations more and at the same time had more difficulty in identifying their feelings than patients with no history of psychiatric disorder. Measure of counter-dependency was not able to differentiate chronic pain patients from healthy controls.

discussion

In our chronic pain patient sample, alexithymia was significantly higher than in healthy controls. Chronic pain patients had more difficulty in identifying feelings and distinguishing them from body sensations of emotion. Previous studies have found elevated alexithymia among patients with chronic or persistent pain. From 33% to 53% of patients with various types of persistent pain appear to be alexithymic.^{12,13} We found that ten out of 30 patients (33%) in our sample had high TAS-20 scores for alexithymia and this number is similar to one reported in a recent study on patients with chronic myofascial pain.²³ Alexithymia is thought to impede successful regulation of emotions, particularly negative affects, resulting in chronic sympathetic hyperarousal, physiological sensations, somatosensory amplification, and complaints of physical symptoms.⁸ At least two studies of healthy people found that alexithymia was positively correlated with reported pain during experimental pain induction or during medical procedures.^{24,25} In these studies, alexithymia was inversely related to pain tolerance and, to a lesser extent,

to pain threshold. These results suggest that alexithymia is associated with enhanced sensitivity not only to internal (somatic) sensations, but also to externally induced pain.²⁴ Lumley *et al.*²³ in a sample of 80 chronic myofascial pain patients found that alexithymia was correlated with the affective and unpleasantness component of pain. The emotional regulation deficits in alexithymic subjects may lead to depression, which appears to mediate alexithymia's relationship to affective pain. Recent research by Kosturek *et al.*⁵ found that, when depression was taken into account, alexithymia became unrelated to chronic pain. It is still unclear whether alexithymia is associated only with the presence of chronic pain *per se*, or whether it is also associated with the severity of pain and disability among people with pain.²³ It is a matter of debate whether alexithymia characterizes some chronic pain populations^{11,12,14} or whether it is more a function of comorbid psychopathology than chronic pain *per se*.^{5,6,13} Though our findings indicate elevated levels of alexithymia in chronic pain populations, they do not address the issue of comorbid psychopathology as a confounding factor. An interesting finding of ours is that only difficulty in identifying feelings subscale of TAS-20 contributed to the difference in alexithymia measure. Difficulties in the ability to identify and differentiate emotions and somatic experiences are core features of the alexithymic construct. Unable to use affects as signals of inner psychic events, many alexithymic individuals are thought to focus on, and to amplify, the somatic sensations of emotional arousal, which are then experienced as overwhelming somatic distress and/or misinterpreted as signs of disease.⁹ Chronic pain patients with a history of psychiatric disorders scored higher on alexithymia than those without a history of psychiatric disorders. We suggest that psychological distress may mediate the relationship between alexithymia and chronic pain. Chronic pain is often comorbid with depression and alexithymia is also substantially related to depression and may predispose to it. Though no actual measurement of psychological distress has been made in our study we think that past psychiatric disorder may also predispose individuals to alexithymia, bearing in mind the already established connection between negative affectivity and alexithymia.

In our study, chronic pain patients showed significantly higher levels of somatosensory amplification. There is great variability among individuals in sensitivity to visceral or somatic sensation. The threshold and tolerance for pathological pain varies greatly among individuals.⁴ Our study reveals that, at least in our chronic pain sample, patients experience their bodily sensations as intense, noxious and disturbing. Amplification appears to have both trait-like and state-like properties.⁴ Although a causal link is hard to draw from these data because of its cross-sectional nature, we may think that those individuals who self-scrutinize, who have a tendency to select and focus on relatively weak or infrequent sensations and those who have a tendency to appraise visceral and somatic sensations as abnormal (namely the main elements of somatosensory amplification) might be more prone to chronic pain. In the study by Kosturek *et al.*⁵ a controlled study was undertaken to assess alexithymia and somatic amplification among 50 medical outpatients with chronic pain referred for psychiatric consultation. Data analysis revealed low scores on the TAS-20 and SSAS for the pain patients, compared with a control group without pain. In this sample, depression and anxiety were the primary determinants of alexithymia and somatic amplification, rather than pain. In another study,²⁶ somatosensory amplification was found to be higher in patients with a history of myofascial pain. These processes may underlie a tendency to express distress in somatic rather than affective terms, leading to somatized or masked depression. Myofascial pain subjects and con-

trols differed significantly on measure of somatosensory amplification. History of depression or current psychological distress did not account for group differences.²⁶ A study by Gregory *et al.*⁶ did not find significant difference between chronic pain subjects and healthy controls in terms of somatosensory amplification. However, somatosensory amplification scores were significantly higher in subjects having pain involving the head, chest, abdomen, or pelvis than in subjects having pain only in their backs or extremities. In our sample, chronic pain patients with a prior history of psychiatric disorder had significantly higher somatosensory amplification scores than those patients without a prior psychiatric disorder. This is in line with the finding of Kosturek *et al.*⁵ where depression and anxiety were primary determinants of somatosensory amplification scores. Spinhoven and van der Does²⁷ suggest that self-reported somatosensory amplification only modestly contributes to the general process of somatization independent of the level of anxiety and depression. Barsky⁴ draws attention to the role of anxiety and depression in introducing a negative bias into the cognitive assessment of an individual's health. It remains unclear to what extent the SSAS captures an underlying mediating process of symptom amplification. In two studies with university students, Aronson *et al.*²⁸ found that the SSAS correlated with measures of symptom reporting and with several indices of general distress, including anxious and depressive symptoms and negative emotionality. The authors conclude that the SSAS is more likely an index of negative emotionality and general distress than a valid measure of somatic sensitivity. On the contrary, a study where psychiatric outpatients were recruited showed that SSAS scores for somatosensory amplification were significantly associated with SCL-90 somatization scores, independently of gender, presence of physical disorder, and level of anxiety and depression.²⁸ We did not assess levels of anxiety and depression in our study. What we can say from our data is that past psychiatric distress might have contributed to somatosensory amplification in this particular subset of patients.

Our findings differ from the findings of two previous studies^{6,17} in delineating the role of counter-dependency in chronic pain patients. The authors of these studies, after finding elevated levels of counter-dependency in their chronic pain samples, concluded that self-reliant and hard-working individuals who minimize distress are less likely to seek rest and medical attention after an acute injury, thereby increasing their chances for developing a chronic condition.⁶ An interesting finding of this study is that subjects with pain exclusively in the back and/or extremities were likely to have strong counter-dependency traits. Our chronic pain group did not differ from healthy controls in terms of CDS. This finding shows that counter-dependency may not be regarded as a psychological marker in Turkish chronic pain patients. Our negative finding may then be due to cultural differences. The counter-dependency construct reflects the Western ethos of individualism and may not fit well in the context of collectivistic cultures. The validity of this construct has to be studied in non-Western cultures before employing it as an instrument.

Finally, we should acknowledge several limitations of our study. Our study sample is rather small to draw definite conclusions. Chronic pain patients comprise a heterogeneous group and this may explain the wide discrepancy of the results in studies with these patients. We did not assess patients with an anxiety or a depression scale and therefore it was not possible to establish any association between these constructs and our study measures. Given the fact that alexithymia and somatosensory amplification may be influenced by the levels of anxiety, depression or psychological distress,

this is a methodological limitation in our study. Though somatosensory amplification scale and counter-dependency scale were found to be reliable in our study subjects, these scales need to be validated in Turkish population studies. We relied on patient reports on the information about past history of psychiatric disorders; we could rather have interviewed the patients with a more structured instrument like SCID. If the study sample had been larger we would have the opportunity to see the predictors of pain severity in our study group. Despite all shortcomings, to our knowledge this study is the first one assessing the relevance of somatosensory amplification and counter-dependency in a non-Western chronic pain population. This study has to be repeated with a larger study sample, employing different psychological distress measures and a structured interview to probe past and current psychiatric disorders.

In conclusion, chronic pain patients are more alexithymic and they amplify their somatic sensations more than healthy individuals. Chronic pain patients with a prior history of psychiatric disorder also tend to be more alexithymic, more amplifying and older than patients without a psychiatric disorder.

references

1. Feuerstein M, Beattie P, Biobehavioral factors affecting pain and disability in low back pain: mechanisms and assessment, *Phys Ther* **75**, 267–80 (1995).
2. Barsky AJ, Wyshak G, Klerman GL, The somatosensory amplification scale and its relationship to hypochondriasis, *J Psychiat Res* **24**, 323–34 (1990).
3. Barsky AJ, Wyshak G, Hypochondriasis and somatosensory amplification, *Br J Psychiatry* **157**, 404–9 (1990).
4. Barsky AJ, Amplification, somatization and the somatoform disorders, *Psychosomatics* **33**, 28–34 (1992).
5. Kosturek A, Gregory RJ, Sousou AJ, *et al.*, Alexithymia and somatic amplification in chronic pain, *Psychosomatics* **39**, 399–404 (1998).
6. Gregory RJ, Manning J, Berry SL, Pain location and psychological characteristics of patients with chronic pain, *Psychosomatics* **41**, 216–20 (2000).
7. Taylor G, Alexithymia: Concept, measurement, and implications for treatment, *Am J Psychiatry* **141**, 725–32 (1984).
8. Lumley MA, Stettner L, Wehmer F, How are alexithymia and physical illness linked? A review and critique of pathways, *J Psychosom Res* **41**, 505–18 (1996).
9. Taylor GJ, Parker JDA, Bagby MA, *et al.*, Alexithymia and somatic complaints in psychiatric outpatients, *J Psychosom Res* **36**, 417–24 (1992).
10. Wise TN, Mann LS, The attribution of somatic symptoms in psychiatric outpatients, *Compr Psychiatry* **36**, 407–10 (1995).
11. Postone N, Alexithymia in chronic pain patients, *Gen Hosp Psychiatry* **8**, 163–7 (1986).
12. Cox BJ, Kuch K, Parker JDA, *et al.*, Alexithymia in somatoform disorder patients with chronic pain, *J Psychosom Res* **38**, 523–7 (1994).
13. Millard RW, Kinsler BL, Evaluation of constricted affect in chronic pain: an attempt using the Toronto Alexithymia Scale, *Pain* **50**, 287–92 (1992).
14. Lumley MA, Asselin LA, Norman S, Alexithymia in chronic pain patients, *Compr Psychiatry* **38**, 160–5 (1997).
15. Sayar K, Bilen A, Arikian M, Kronik agri hastalarinda ofke, benlik saygisi ve aleksitimi (Anger, self-esteem and alexithymia in chronic pain patients). *Turkiye Klinikleri Psikiyatri Dergisi* **2**, 36–42 (2001).
16. Barsky AJ, Somatoform disorders, in: *Comprehensive Textbook of Psychiatry*, Kaplan HI, Sadock BJ (Eds), Vol. 1, 5th edn, pp. 1009–27. Williams and Wilkins, Baltimore, MD (1989).
17. Gregory RJ, Berry SL, Measuring counterdependency in patients with chronic pain, *Psychosom Med* **61**, 341–5 (1999).
18. Hujkisson HC, Visual analog scales, in: *Handbook of Psychiatric Measures*, pp. 601–3. American Psychiatric Press, Washington, DC (2000).
19. Bagby RM, Parker JDA, Taylor GJ, The 20-item Toronto-Alexithymia-Scale, 1. Item selection and cross-validation of the factor structure, *J Psychosom Res* **38**, 23–32 (1994).
20. Bagby RM, Taylor GJ, Parker JDA, The 20-item Toronto-Alexithymia-Scale, 2. Convergent, discriminant, and concurrent validity, *J Psychosom Res* **38**, 33–40 (1994).
21. Sayar K, Gulec H, Ak I, Yirmi soruluk Toronto Aleksitimi Olcegi'nin guvenirligi ve gecerligi (The reliability and validity of the Twenty-item Toronto Alexithymia scale), in:

- 37 *Ulusal Psikiyatri Kongresi Bilimsel Calismalar Ozet Kitabi (37th National Congress of Psychiatry, Scientific Studies Abstract Book)*, Istanbul (2001).
22. Barsky AJ, Wyshak G, Klerman GL, The Somatosensory Amplification Scale and its relationship to hypochondriasis, *J Psychiatry Res* **24**, 323–34 (1990).
 23. Lumley MA, Smith JA, Longo DJ, The relationship of alexithymia to pain severity and impairment among patients with chronic myofascial pain. Comparisons with self-efficacy, catastrophizing, and depression, *J Psychosom Res* **53**, 823–30 (2002).
 24. Nyklicek I, Vingerhoets A, Alexithymia is associated with low tolerance to experimental painful stimuli, *Pain* **65**, 471–5 (2000).
 25. Putterman E, Byrne N, Ditto B, Alexithymia and symptom reporting following blood donation, *Psychosom Med* **63**, 138–9 (2001).
 26. Raphael KG, Marbach JJ, Gallagher RM, Somatosensory amplification and affective inhibition are elevated in myofascial face pain, *Pain Medicine* **1**, 247–53 (2000).
 27. Spinhoven P, van der Does AJW, Somatization and somatosensory amplification in psychiatric outpatients: An explorative study, *Compr Psychiatry* **38**, 93–7 (1997).
 28. Aronson KR, Barrett LF, Quigley KS, Feeling your body or feeling badly. Evidence for the limited validity of the Somatosensory Amplification Scale as an index of somatic sensitivity, *J Psychosom Res* **51**, 387–94 (2001).