

Physical Components of Depression and Psychomotor Retardation

Yves Lecrubier, M.D.

The role of somatic symptoms in patients with depression has been historically underestimated and underrecognized. Results of the World Health Organization Collaborative Project on psychological problems in general health care have established somatization as a frequently cited feature of depression in patients seen by primary care physicians, and a number of ensuing studies have supported these data. Approximately 73% of patients with depression are affected by lack of energy and fatigue. In addition, patients with depression experience a greater number of somatic symptoms, including back and chest pain, abdominal pain, headache, fatigue, and weakness, than do patients with general anxiety and patients with no psychiatric diagnosis. Additional studies have reported a correlation between depression and psychomotor retardation, indicating that psychomotor retardation comprises a much broader spectrum of depression than was originally thought. To date, this research has focused primarily on the Parkinsonian-like gait and stride observed in patients with depression compared with the normal gait and stride observed among healthy control subjects. In addition, data have indicated significantly improved psychomotor retardation in patients with depression after 3 months of treatment.

(J Clin Psychiatry 2006;67[suppl 6]:23–26)

Depression is a natural reaction of people who have no fight-or-flight capacity in response to an adverse external or internal event that the person either deems unacceptable or is ill-equipped to handle. People naturally spare physical and emotional resources and, in trying to do so, may block or freeze activities, resulting in loss of energy, lethargy and weakness, fatigue, and psychomotor retardation.

Depression has historically been viewed in terms of psychomotor depression, and only recently did the field of psychiatry give priority to mood states and the subjective manifestation of depression. What was once called psychomotor retardation is now considered a mood disorder or major psychomotor disturbance. However, the emotional and physical symptoms of depression as identified by the DSM-IV-TR¹ are not independent of each other (Table 1); rather they are major dimensions of depression that are reflected throughout the mood-disorder spectrum. Additionally, these symptoms contribute to withdrawal or loss of energy, which are secondary dimensions of depression that are often underestimated.

SOMATIC SYMPTOMS OF DEPRESSION

Tylee and Gandhi² have stated that lack of energy and fatigue are as frequent as mood problems in depression, affecting 73% and 76% of patients, respectively. According to Simon et al.,³ results from the World Health Organization's (WHO) study of psychological problems in general health care concur that somatic symptoms of depression are common. The WHO collaborative study screened approximately 25,000 patients at 15 primary care centers in 14 countries. Of these patients, 5447 went on to receive structured assessments of depressive and somatoform disorders. A total of 1146 patients were diagnosed with major depression. The mean number of physical symptoms was higher in patients with depression (5.2) than in patients with no psychiatric diagnosis (2.8), and it was 4.6 in patients with depression and no comorbid physical illness.

Most patients (69%) in the WHO collaborative study consulted their primary physicians not for depressive symptoms but rather for physical symptoms only.⁴ Back pain (35%), headache (34%), and joint pain (25%) were among the top 3 overall reasons for consultation, followed by lethargy, weakness, and dizziness (23%). The highest odds ratio for the risk of depression when somatic symptoms were present was the greatest for weakness/lethargy (2.3) followed by pain (2.0), shortness of breath (1.7), dizziness (1.7), and frequent urination (1.6).⁵ Overall, patients with depression complained of physical symptoms more often than patients without depression (Table 2).

*From the Hôpital La Salpêtrière, INSERM, Paris, France.
This article is derived from the planning roundtable
"The Role of Dopamine and Norepinephrine in Depression
and Antidepressant Treatment," which was held July 22, 2005,
in Taplow, Berkshire, U.K., and supported by an educational
grant from GlaxoSmithKline.*

*Corresponding author and reprints: Yves Lecrubier, M.D.,
47 Bd de l'Hôpital, 75651 Paris cedex 13, France.*

Table 1. Spectrum of Symptoms in Depression^a

Emotional Symptoms	Physical Symptoms
Sadness and tearfulness	Tiredness/fatigue
Loss of interest	Sleep disturbances
Anxiety/irritability	Headaches
Hopelessness	Psychomotor
Concentration difficulties	Gastrointestinal disturbances
Guilt	Appetite changes
Suicidal ideation	Body aches and pains

^aAdapted from DSM-IV-TR.¹

According to a number of current theories on depression, patients who present with physical expressions may be unable to express their psychological state adequately; therefore, the physical symptoms could either be a “real” part of depression or a derivative of the nonexpression of a psychological problem. Approximately one third of patients with a physical illness are depressed,⁶ which in turn affects the presence of somatic symptoms.

The data from the WHO collaborative study represents what the patients were saying (i.e., the reasons why they were coming to their doctors and spontaneously complaining) rather than their responses to an interviewer as to whether or not a particular symptom was present, as was previously illustrated. The data show a number of constants across the 14 different countries: the number of depressed patients who spontaneously complained at the psychological level was low (25% to 30% maximum), and the most common complaint was pain, followed by weakness and fatigue.⁴ The percentage of patients with depression who were experiencing weakness (12%) did not differ greatly from the number of patients experiencing depressive symptoms (15%; see Table 2), and there was much more specificity for these 2 symptoms than for pain, which was consistent with results observed when symptoms were recorded by an interviewer.

The WHO collaborative study also examined variations in the probability of somatized depression. Indeed, in primary care, the existence of somatic symptoms does predict the existence of a major depressive episode with odds ratios of about 2.5 for 2 to 3 somatic symptoms and > 10 for more than 5 symptoms. As reported by Simon et al.,⁷ denial of psychological symptoms was relatively low regardless of center locale, ranging from 2% to 26%. Somatic presentation (spontaneous complaints) was variable from one center to another, with significant differences between Paris at 45% and Ankara at 95% ($p = .002$). These differences indicate a cultural relationship. On the other hand, no significant difference among centers was found in unexplained somatic symptoms (interviewer questions), which ranged from 30% to 60%. These data suggest a strong relationship between somatic symptoms and depressive symptoms. Although a similar number of physical symptoms are present, some cultures express more complaints than others. When one looks at the psychological versus somatic

Table 2. Spontaneous Symptom Complaints in Patients With Depression Versus Patients Without Depression^a

Symptom	Patients With Depression (%)	Patients Without Depression (%)
Back/chest pain	16	13
Depressive	15	2
Anxiety	15	3
Other pain	13	15
Abdominal pain	13	11
Headache	12	9
Weakness	12	6
Genito-urinary	10	7
Cough/cold	10	15
Sleep problems	7	3
Vertigo	7	5
Fever	2	4
Weight loss	2	1
Other somatic < 2% each	24	27

^aData from Lecrubier.⁵

complaints of these patients, patients with a low level of complaints of psychological symptoms may be expected to have a high number of complaints of physical symptoms and vice versa. However, results of the WHO collaborative study indicated the opposite: patients with a low level of psychological complaints had a low level of physical complaints (e.g., Japan or China), and a large number of psychological complaints was equated with a large number of physical complaints (e.g., Chile or Brazil). Therefore, the tendency to complain at both a physical and psychological level is culturally based, but no balance between physical and psychological symptoms has been observed. Overall, these data strongly suggest that physical symptoms are part of the depression symptomatology.

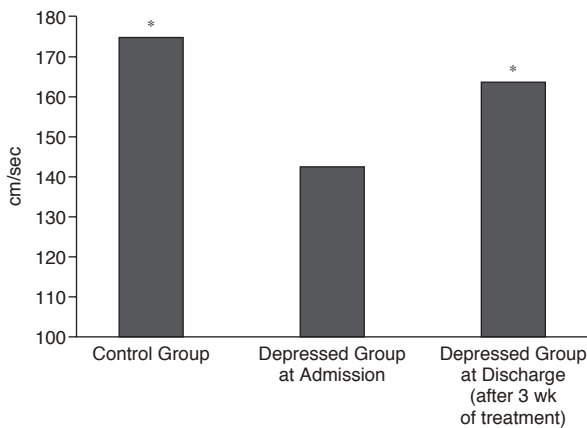
DEPRESSION AND PSYCHOMOTOR RETARDATION

As indicated by the WHO collaborative study, somatic symptoms are a real component of depression. Traditionally, the roles of these symptoms—including psychomotor retardation, weakness, and lethargy—have been underestimated. A group was established to test the correlation of psychomotor retardation, weakness, and lethargy among a number of factors, ranging from gait and stride to language and verbal flow to memory, concentration, and interest in usual activities, in a sample of 150 patients with depression.⁸ Results indicated that almost 60% of variance found in Depressive Retardation Rating Scale scores could be explained by the psychomotor retardation factor alone. These data support the concept that psychomotor retardation is a much broader symptom of depression than was originally thought.⁹

GAIT AND STRIDE STUDIES

To illustrate this point, the gait patterns of 26 patients with depression were analyzed along with those of 26

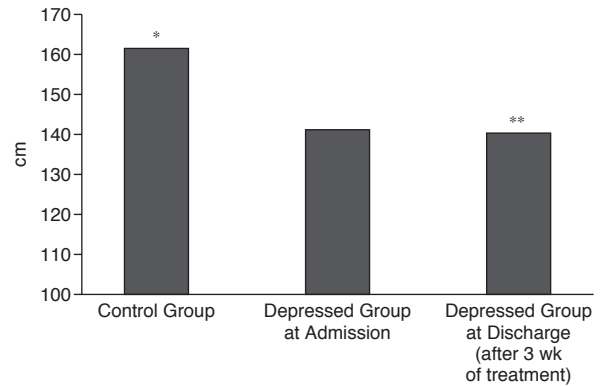
Figure 1. Heel Propulsion Velocity Among Depressed Patients and Healthy Control Subjects^a



^aData from Hergueta et al.¹⁰

* $p < .001$ vs. depressed group at admission.

Figure 2. Length of Stride Among Depressed Patients and Healthy Control Subjects^a



^aData from Hergueta et al.¹⁰

* $p < .01$ vs. depressed group at admission.

** $p < .001$ vs. control group.

healthy control subjects.¹⁰ Patients were asked to walk toward the researcher and were filmed walking perpendicular to a camera. To record their natural walk, patients were not informed when they were or were not being filmed. The recordings were then computerized and analyzed; heel propulsion velocity (Figure 1) and the length of the stride (Figure 2) were the 2 main variables that were examined. Patients with depression had a heel propulsion velocity approximately 19% lower than that in healthy control subjects (groups were matched for age, height, and sex). When patients with depression were discharged after 3 weeks of treatment, heel propulsion velocity returned to normal, indicating the correlation between psychomotor retardation and depression. A difference in length of stride also was observed between patients with depression and healthy controls. Patients with depression had a shorter length of stride, resembling a Parkinsonian-like stride, than did healthy control subjects. However, upon discharge after 3 weeks of treatment, the length of stride remained shortened in patients with depression. The combined heel propulsion velocity and length of stride data reveal that not all psychomotor retardation improves at the same rate.

An additional study¹¹ was conducted in which both heel propulsion velocity and length of stride were measured in depressed patients treated with venlafaxine. Results were evaluated after 3 weeks, 4 weeks, and 3 months of treatment to see whether length of stride improved. Researchers found that speed improved early in the study (i.e., by week 2 or 3) and continued to improve throughout the 3 months. The length of stride did not significantly improve in the initial weeks of treatment, but it did significantly improve after 3 months of treatment. The researchers theorized that if speed is related to serotonin structures and length of stride is related to the dopaminergic dimension,

changes in dopaminergic structures over the long term with antidepressant medications led to the long-term improvements in the length of stride.

CONCLUSION

The significance and prevalence of somatic symptoms in depression has been established; however, the role of psychomotor retardation in depression is just beginning to be understood by researchers. Studies analyzing the gait and stride of patients both with and without depression have demonstrated a slower, Parkinsonian-like stride in patients with depression compared with that in healthy control subjects. Additional data have shown significant improvement in length and speed of stride in patients with depression after receiving 3 months of antidepressant treatment.

REVIEW QUESTION

In your practice, do you address physical symptoms and psychomotor retardation when planning a treatment regimen for a depressed patient?

Drug name: venlafaxine (Effexor).

Disclosure of off-label usage: The author has determined that, to the best of his knowledge, no investigational information about pharmaceutical agents that is outside U.S. Food and Drug Administration–approved labeling has been presented in this article.

REFERENCES

1. American Psychiatric Association. Diagnostic and Statistical Manual, Fourth Edition, Text Revision. Washington, DC: American Psychiatric Association; 2000.

- Association; 2000
2. Tylee A, Gandhi P. The importance of somatic symptoms in depression in primary care. *Prim Care Companion J Clin Psychiatry* 2005;7:167–176
 3. Simon GE, VonKorff M, Piccinelli M, et al. An international study of the relation between somatic symptoms and depression. *N Engl J Med* 1999;341:1329–1335
 4. Üstün TB, Von Korff M. Primary mental health services: access and provision of care. In: Üstün TB, Sartorius N, eds. *Mental Illness in General Health Care: An International Study*. Chichester, England: Wiley Press; 1995:347–360
 5. Lecrubier Y. Dépression et symptômes somatiques. *Nervure* 2005; 18(Sept, special issue):11–14
 6. Katon W, Sullivan MD. Depression and chronic medical illness. *J Clin Psychiatry* 1990;51(6, suppl):3–11
 7. Simon G, Lecrubier Y, Üstün TB. Somatic symptoms and psychiatric disorder: an international primary care study. *Rev Contemp Pharmacother* 1996;7:279–285
 8. Widlocher DJ. Psychomotor retardation: clinical, theoretical, and psychometric aspects. *Psychiatr Clin North Am* 1983;6:27–40
 9. Jouvent R, Lecrubier Y, Stoleru L, et al. Analyse multifactorielle de l'échelle de ralentissement dépressif utilisée chez les déprimés ambulatoires. *Psychologie Médicale* 1981;13:97–107
 10. Hergueta T, Delgado F, Lecrubier Y. Quantitative video-analysis of gait in depressed inpatients [abstract]. *Eur Neuropsychopharmacology* 1996; 6(suppl 4):91
 11. Hergueta T, Lecrubier Y. Specific pattern of gait in depression: state and trait markers. Presented at the European Winter Conference on Brain Research; Mar 6–13, 1999; La Plagne, France