

Medicographia

Major Issues in Depression

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Motor activity, posture, and facial expression have revealed mood in human beings since the earliest civilizations. The psychiatrist Karl Kleist (1879-1960) pointed out that psychomotor activity also implies subjective experiences. He assigned these experiences to the frontal lobe and the psychomotor performance to the basal nuclei.¹ Sigmund Freud (1856-1939) attempted to associate neurochemical issues with instinctual drives as a way of understanding the individual human unconscious/conscious mind. Instinctual patterns impose far more demands on the nervous system than external stimuli. They force it to engage in complex activities, and, without these emotional and perceptual feelings, all that is related to the mental representation aspects fails. Excitement flows, the affective tone does not take place, and the limited psychic inscription cannot stop quantitative transmission of drive.² Breakthroughs in neuroscience demonstrated that the prefrontal cortex (anterior and inferior frontal lobe, area 8) is involved in the planning, initiation, and sequence of voluntary movements. This requires the drive and initiative to execute that which is has been thought or planned. Motivation is mediated through the cortex frontal-orbital pathway (limbic areas), which can inhibit the prefrontal areas or liberate frontal areas and generate disorders in the frontal-basal pathways (promoting passive or aggressive behaviors). Movement depends on the continuous inflow of information from afferent sensory connections to prefrontal areas. If this information becomes unnecessary, it extinguishes by inhibition of frontal-orbital impulses.³ Psychomotor retardation, or generalized slowing down of body movements (motility, attitude, and impairments of speech), is perceived by the patient. Inhibition, loss of interest and lack of pleasure (anhedonia) are diagnostic of major depressive illness (Diagnostic and Statistical Manual of Mental Disorders, 4th Edition [DSM-IV]). There is no evidence to date of the existence of biological markers of psy-

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Psychomotor retardation in depressive patients in spite of the fact that it involves the disturbance of several neurotransmitters, especially those belonging to the noradrenergic and dopaminergic systems. Plasma arginine-vasopressin concentrations are low in depressive patients with daytime psychomotor retardation, and elevated in those who experience increased motor activity during sleep.⁴ Likewise, growth hormone pulses are lower in female patients with major depressive illness associated with higher scores on the retardation item of the Hamilton Depression Rating Scale (HDRS).⁵ Studies on fluoxetine show that it is more efficient in inhibited depressive illness in patients with low scores for psychomotor retardation, which suggests that the serotonin system may play a secondary role.⁶ Psychomotor retardation may be the most consistent predictor of good response to tricyclic antidepressants and indicate the onset of improved social functioning.⁷ The newer antidepressant agents elicit a more rapid disinhibition of motor retardation, and it proved necessary to develop scales to reveal this phenomenon. Psychomotor retardation improves slowly with antidepressive treatment. Although the professional's clinical impression is based on the physical appearance and gestures of the depressive patient, these are unreliable as medical evidence in short- and mid-term clinical trials. Parametric assessment to evidence the patient's subjective impressions emerging at interview is a way to objectively document impressions and confirm the improvement or worsening under pharmacological treatment. The Wildöcher scale and the Depressive Retardation Rating Scale have shown their versatility in documenting and correlating psychomotor retardation with the anhedonia of these patients. In view of the regularly observed alterations in neurotransmission mechanisms, it would be important to correlate these types of scales with negative symptoms of schizo-affective and/or schizophrenic patients.⁸⁻¹⁰ □