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# Anosognosia in Tardive Dyskinesia: "Tardive Dysmentia" or "Tardive Dementia"?

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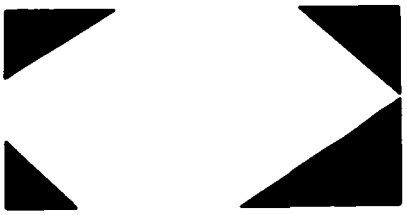
## At Issue




### Abstract

**Wilson et al. (1983) called attention to the increasing incidence of affective symptomatology in schizophrenic patients on chronic neuroleptic medication. They designated the emerging syndrome as tardive dysmentia in order to emphasize its connection with the pathophysiology of tardive dyskinesia. The present contribution suggests that the emotional indifference or frank anosognosia of abnormal involuntary movements noted in the great majority (95 percent) of patients with tardive dyskinesia is another possible feature of tardive dysmentia. An attempt was made to explain anosognosia, and some difficulties that mar interpretation of data were pointed out. Anosognosia was proposed to represent a symptom related to some sort of cognitive disorder accompanying tardive dyskinesia, at least in the elderly. It was suggested that dementia may be associated with a predominant impairment of dopaminergic circuitry in the right cerebral hemisphere.**


Several investigators have noted a significant inverse correlation between the severity of dyskinesia and depressive symptomatology (Vogel 1982; Oyewumi et al. 1983). Wilson et al. (1983) proposed that the insidious intrusion of affective symptomatology in chronic medicated schizophrenics is a behavioral equivalent of tardive dyskinesia, which they provocatively christened "tardive dysmentia."

While interviewing institutionalized patients with tardive dyskinesia (Holden, Sandler, and Myslobodsky 1984; Myslobodsky, Holden, and Sandler 1984), I was struck by the fact that most of them (95 percent) were oblivious to their faciolingual dyskinesia (see table 1 for characteristics of the study sample). I had imagined that at least patients who had Parkinson's signs concurrently with tardive dyskinesia would express concern when asked directly about their involuntary movements. However, they too appeared to be indifferent to their abnormality, even when it was very severe. This lack of awareness may explain the fact that even grotesque dyskinesia does not encourage social withdrawal and, as Owen (1979) has emphasized, is a distressing condition mostly for the patient's family. Our informal observations suggest that minimization or anosognosia adds another feature to the concept of tardive dysmentia inasmuch as our patients on chronic neuroleptic medication manifested other affective symptoms akin to those described in "dysmentia."

Matters are complicated by the fact that the major symptoms of tardive dysmentia outlined by Wilson et al. (1983)—for example, euphoria; emotional incontinence; impaired control of impulses; episodes of petulance, irritability, withdrawal, and apathy; loquaciousness; and verbal stereotypy—may accompany a moderate state of dementia, with or without concomitant abnormal involuntary movements (Blass 1982; Olkinuora 1982; Benson 1984). Moreover, the course of tardive dyskinesia is most likely to be severe in the elderly or in individuals with organic brain disorder (Chouinard et al. 1979). In computed tomography studies, Bartels and Themelis (1983) recently demonstrated structural abnormalities in the basal ganglia of

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**Table 1. Characteristics of the study sample**

| Group | Age | | Sex (M/F) | Mean TD score[1] | Mean Parkinson's score[2] | Mean current dose[3] (mg/day) | Years of hospitalization | |
|---|---|---|---|---|---|---|---|---|
| | Mean | Range | | | | | Mean | Range |
| Schizophrenics[4] ($n$ = 23) | 63.3 | 38–88 | 12/11 | 3.7 | 2.1 | 872 | 28.9 | 4–60 |
| Manic-depressives ($n$ = 7) | 67.7 | 55–83 | 3/4 | 3.6 | 1.1 | 228 | 228 | 3–50 |
| Epilepsy ($n$ = 5) | 60 | 49–70 | 2/3 | 3.6 | 0.8 | 360 | 30.6 | 15–45 |
| OBS[5] ($n$ = 14) | 61 | 37–84 | 8/6 | 3.6 | 1.9 | 184 | 13.2 | 2–60 |

[1] Mean number of items on Rockland scale (Simpson et al. 1979).
[2] Mean number of items on Simpson and Angus (1970) scale.
[3] Chlorpromazine equivalent units.
[4] All subtypes.
[5] OBS = organic brain syndrome with psychotic symptoms, agitation, and assaultiveness.

patients with tardive dyskinesia. A large number of residents in homes for the elderly without a history of neuroleptic treatment reportedly exhibit abnormal involuntary movements (Bourgeois et al. 1980; Blowers 1981). Finally, there is an intriguing overlap between the neurotransmitter systems implicated in dementia (Gottfries 1982; Reisberg, Ferris, and Gershon 1983) and those assumed to malfunction in tardive dyskinesia (Klawans 1973). Similar to dementias, tardive dyskinesia may be considered a case of overmedication of the elderly (Cahn and Diesfeldt 1973), in which the fragile balance between neurotransmitter systems controlling locomotion and arousal is disrupted. Since aging itself confers a significant risk of both tardive dyskinesia and dementia, it is uncertain whether tardive dysmentia is a distinct syndrome or an unrecognized case of "tardive dementia."

Why patients with tardive dyskinesia should develop signs of emotional indifference is not immediately apparent. A number of investigators—most notably, Alexopoulos (1979), Rosen et al. (1982), Simpson (1975), and Smith et al. (1979)—have written on the subject, but no explanation has yet emerged. In fact, there may be more than a single explanation of this phenomenon. Some of the problems in interpreting anosognosia may also be relevant to an understanding of tardive dysmentia.

Wilson et al. (1983) have proposed that tardive dysmentia, similar to tardive dyskinesia (Klawans 1973), is associated with supersensitive dopamine receptors. If dopamine-mediated activity contributes significantly to reward and emotional behaviors, dopamine hyperactivity might be expected to promote an intrusion of affective symptomatology and enhance the "hedonic tone" of schizophrenia. The major limitation of this explanation is that the hyperproduction of either $D_1$ or $D_2$ receptors in tardive dyskinesia has yet to be supported experimentally (Hyttel, Christensen, and Arnt 1983). Also, the dopamine hypothesis of schizophrenia, which postulates an overactivity in the mesolimbic system, does not explain the profound anhedonia associated with this psychosis (Meehl 1962; Rado 1962).

Antiparkinson medication might be perceived as another factor of mood improvement. Unfortunately, in the study by Wilson et al. (1983), no data were presented relating to this variable. However, among patients with tardive dyskinesia examined by us, only a fraction received antiparkinson drugs (Holden, Sandler, and Myslobodsky 1984). Also, in a carefully controlled study, Mindham et al. (1972) failed to support claims of mood elevation in patients on amantadine and orphenadine. Even dysphoric reaction in patients with early akineto-rigid symptomatology were not alleviated by antiparkinson medication (Singh and Kay 1979).

Minimization and indifference might reflect a general disturbance of body image and self-identity in schizophrenia (Fisher and Cleveland 1958). However, tardive dyskinesia is not limited to schizophrenia. Some of our manic and epileptic patients showed most convincing examples of

unawareness. Hence, the above variable would not fully account for this feature of tardive dyskinesia.

Minimization and denial of disease are common features in the epilepsies (De Angelis and Vizioli 1983). Schizophrenic patients often have epileptiform abnormalities of the electroencephalogram (EEG) (e.g., Stevens and Livermore 1982) that are not necessarily detectable by scalp electrodes. These seizure potentials may conceivably act as a "mild" chronic electroconvulsive therapy, postulated by De Angelis and Vizioli to improve mood in epileptic patients. While this possibility warrants a special investigation, it should be recalled that the so-called B-mitten pattern remains the only EEG correlate of tardive dyskinesia described so far (Wegner et al. 1979). However, the prevalence of B-mittens was shown to decline from age 40 upward, so that in patients older than 40 (i.e., in the majority of patients with tardive dyskinesia) no mittens are recorded. Also, it is of interest that B-mittens were originally believed to correlate with the Parkinson's syndrome (Winfield and Sparer 1954), which is seldom associated with epilepsy.

Another possibility reinforces the contention that tardive dyskinesia appears upon a background of neuroleptic-induced parkinsonism. It proposes that the symptoms in question are not idiosyncratic ones but originate along the akinesia/hyperkinesia dividing lines of parkinsonism. Parkinson's patients affected by tremor, which is considered a transitional symptom between parkinsonism and hyperkinesia (Chouinard et al. 1979), feel subjectively better than patients who have hypokinesia as a leading sign (Vogel 1982). Similarly, individuals treated with L-dopa are reportedly better able to tolerate severe dystonic and choreiform hyperkinesis than being hypokinetic (Duvoisin 1982; Vogel 1982). Duvoisin has commented that a patient's preference may represent a useful guide to dosage in cases of poor response to long-term L-dopa treatment. We (Holden and Myslobodsky, unpublished) elicited bitter complaints of stiffness and inability to walk from patients with tardive dyskinesia only within 2 weeks of cessation of anticholinergic medication. However, there was no appreciable difference in the degree of minimization among patients with various symptoms of parkinsonism, including rigidity, as far as faciolingual symptomatology was concerned.

Like Wilson et al. (1983), we were struck by the less socially disruptive character of psychotic patients with tardive dyskinesia. It is tempting to liken this perplexing phenomenon to the anosognosia of right-hemisphere-damaged patients. Although there is no indication of asymmetric brain involvement in tardive dyskinesia, such a possibility cannot be ruled out. Some investigators (Chouinard and Jones 1978; Kapit 1978) have hypothesized that the presumed hyperdopaminergia underlying schizophrenia is the result of an initial dopamine deficiency. The fact that extrapyramidal symptomatology occurs in some 40 percent of patients on neuroleptics (Marsden, Tarsy, and Baldessarini 1973) is consistent with this conjecture. Neuroleptic-induced parkinsonism is known to develop against the background of striatal dopamine depletion in a certain population of susceptible individuals (Rajput et al. 1982). It has been proposed that schizophrenia is associated with left hemisphere dysfunction (Flor-Henry 1969, 1983). In keeping with this concept, Waziri (1980) found that peripheral signs of tardive dyskinesia predominate on the right side of the body. While the faciolingual signs of tardive dyskinesia are mostly symmetrical (or, rather, their asymmetries are difficult to establish), certain indications of speech impairment accompany dyskinesia (Darley, Aronson, and Brown 1975). Also, symmetrical faciolingual signs may be tentatively attributed to an abnormality of lateralized mechanisms as the left hemisphere has been implicated in the bilateral control of the facial musculature (Kimura 1982).

The foregoing formed a basis for a neuropsychological study conducted in patients with tardive dyskinesia (Myslobodsky et al. 1985). This study encountered a problem at its inception. Only 18 (about 23 percent) of 79 patients drawn from two hospitals were free of serious cognitive deficits as measured by the Mini-Mental State Examination (Folstein, Folstein, and McHugh 1975). Even among these selected patients, several scored uncomfortably close to 25—the cutoff point between normality and cognitive impairment used by Folstein, Folstein, and McHugh (1975). In the majority of tests, the neuropsychological test performance of the 18 patients with tardive dyskinesia was not inferior to that of similarly screened psychotic controls matched for age, sex, and medication. In the Picture Recall Test, however, patients with tardive dyskinesia performed reliably worse than controls when the target stimuli were positioned in the right hemispatial field. Struve and Willner (1983) have reported impaired performance of patients with tardive dyskinesia on the Conceptual Level Analogy Test, a finding that is in keeping with left hemisphere involvement.

We have already alluded to the suggestive evidence of left hemisphere dysfunction in tardive

dyskinesia offered by Waziri (1980). Unfortunately, his findings of right-sided dyskinesias were based on a small sample and were not replicated in a prevalence study (Myslobodsky, Holden, and Sandler 1984). Also, Jeste and Wyatt (1982) argue that unlike L-dopa-induced hyperkinesias, lateralized tardive dyskinesia is a rather uncommon phenomenon. Finally, P. Taylor (personal communication) has recently found asymmetrical extrapyramidal symptoms in 3 of 12 patients with tardive dyskinesia. In agreement with our own study (Myslobodsky, Holden, and Sandler 1984), all had tremor.

Hence, the hypothesis of selective left hemisphere deterioration in tardive dyskinesia does not find consistent support. Nevertheless, there are some heuristic benefits in entertaining the idea of a lateralized brain abnormality underlying emotional indifference. It is conceivable that cognitive deficits in tardive dyskinesia develop secondarily to right hemisphere abnormality.

This alternative conjecture is based on a number of findings suggesting that functions controlled by the right hemisphere deteriorate more noticeably with advancing years (e.g., Lapidot 1983). However, the contribution of the right hemisphere to the cognitive decline may be difficult to demonstrate experimentally because of the bilateral control of the right hemisphere over orienting reaction-electrodermal responsiveness (Heilman, Schwartz, and Watson 1978; Zoccolatti, Scabini, and Violani 1982; Mintz and Myslobodsky 1983), attention (Mesulam et al. 1976), touch exploration of extrapersonal space (Desmedt 1977), affect (Gainotti 1972, 1983), and some paralinguistic aspects of communication (Weintraub, Mesulum, and Kramer 1981). Even disprosody in patients with tardive dyskinesia (Darley, Aronson, and Brown 1975) may be associated with right hemisphere dysfunction. Hence, inconsistency of attention and incomplete monitoring of changes in various segments of extrapersonal space such as breakdowns of the body image and oscillations in cognitive and emotional reactivity (i.e., phenomena underlying various patterns of the dementias) may conceivably follow pharmacologically induced incapacity of the right hemisphere. It is intriguing that tardive dyskinesia assumes a notoriously severe course in patients with affective psychoses (e.g., Wolf, Ryan, and Mosnaim 1982) which are believed to be associated with right hemisphere abnormalities (Flor-Henry 1969, 1983). Therefore, the last and more prudent possibility relates anosognosia in patients with tardive dyskinesia to some form of cognitive decline associated with dementia disorder, apparently owing to some neuroleptic-induced deficiency within the dopaminergic circuitry.

This article cannot provide a confident answer to the question posed by its title. The problem of tardive dysmentia, as Wilson et al. have correctly pointed out, is that of the behavioral equivalents of tardive dyskinesia have yet to be established. Tardive dyskinesia can be encountered in children and adolescents (Gualtieri et al. 1984) who would hardly be expected to show signs of dementia. Whether they also manifest symptoms of tardive dysmentia or indifference and anosognosia remains to be elucidated.

## References

Alexopoulos, G.S. Lack of complaints in schizophrenics with tardive dyskinesia. *Journal of Nervous and Mental Disease*, 167:125–127, 1979.

Bartels, M., and Themelis, J. Computerized tomography in tardive dyskinesia: Evidence of structural abnormalities in the basal ganglia system. *Archives of Psychiatry and Neurological Sciences*, 233:371–379, 1983.

Benson, D.F. Parkinsonian dementia: Cortical or subcortical? In: Hassler, R.G., and Christ, J.F., eds. *Advances in Neurology*. Vol. 40. New York: Raven Press, 1984. pp. 235–240.

Blass, J.P. Dementia. *Medical Clinics of North America*, 66:1143–1160, 1982.

Blowers, A.J. Epidemiology of tardive dyskinesia in the elderly. *Neuropharmacology*, 20:1339–1340, 1981.

Bourgeois, M.; Bouilh, P.; Tignol, J.; and Yesavage, J. Spontaneous dyskinesias vs. neuroleptic-induced dyskinesias in 270 elderly subjects. *Journal of Nervous and Mental Disease*, 168:177–178, 1980.

Cahn, L.A., and Diesfeld, H.F.A. The use of neuroleptics in the treatment of dementia. *Psychiatrie, Neurologie und Neurochirurgie*, 76:411–420, 1973.

Chouinard, G.; Annable, L.; Ross-Chouinard, A.; and Nestoros, J.N. Factors related to tardive dyskinesia. *American Journal of Psychiatry*, 136:79–83, 1979.

Chouinard, G., and Jones, B.D. Schizophrenia as dopamine-deficiency disease. *Lancet*, II:99–100, 1978.

Darley, F.; Aronson, A.; and Brown, J. *Motor Speech Disorders*. Philadelphia: W.B. Saunders, 1975.

De Angelis, G., and Vizioli, R. Epilepsy and depression. In:

Parsonage, M.; Grant, R.H.E.; Craig, A.G.; and Ward, A.A., eds. *Advances in Epileptology*. New York: Raven Press, 1983. pp. 203-206.

Desmedt, J.E. Active touch exploration of extrapersonal space elicits specific electrogenesis in the right cerebral hemisphere of intact right-handed man. *Proceedings of the National Academy of Sciences of the United States of America*, 74:4037-4040, 1977.

Duvoisin, R.C. Management of patients who fail to respond to levodopa therapy. *Clinical Neuropharmacology*, 5 (Suppl. 1):s13-s18, 1982.

Fisher, S., and Cleveland, S.E. *Body Image and Personality*. Princeton, NJ: Van Nostrand, 1958.

Flor-Henry, P. Psychosis and temporal lobe epilepsy—A controlled investigation. *Epilepsia*, 10:363-395, 1969.

Flor-Henry, P. Hemisyndrome of temporal lobe epilepsy: Review of evidence relating psychopathologial manifestations in epilepsy to right- and left-sided epilepsy. In: Myslobodsky, M., ed. *Hemisyndromes: Psychobiology, Neurology, Psychiatry*. New York: Academic Press, 1983. pp. 149-174.

Folstein, M.F.; Folstein, S.E.; and McHugh, P.R.. Mini-mental state: A practical method for grading the cognitive state of patients for clinicians. *Journal of Psychiatric Research*, 12:189-198, 1975.

Gainotti, G. Emotional behaviour and hemispheric side of lesion. *Cortex*, 8:41-55, 1972.

Gainotti, G. Laterality of affect: The emotional behavior of right- and left-brain-damaged patients. In: Myslobodsky, M.S., ed. *Hemisyndromes: Psychobiology, Neurology, Psychiatry*. New York: Academic Press, 1983. pp. 175-192.

Gottfries, C.G. The metabolism of some neurotransmitters in aging and dementia disorders. *Gerontology*, 28(Suppl. 2):11-19, 1982.

Gualtieri, C.T.; Quade, D.; Hicks, R.E.; Mayo, J.P.; and Schroeder, S.R. Tardive dyskinesia and other clinical consequences of neuroleptic treatment in children and adolescents. *American Journal of Psychiatry*, 141:20-23, 1984.

Heilman, K.M.; Schwartz, H.D.; and Watson, R.T. Hypoarousal in patients with the neglect syndrome and emotional indifference. *Neurology*, 28:229-232, 1978.

Holden, T.; Sandler, R.; and Myslobodsky, M. Tardive dyskinesia: Prevalence and subtypes at Valkenberg Hospital. *South African Medical Journal*, 66:132-134, 1984.

Hyttel, J.; Christensen, A.V.; and Arnt, J. Neuroleptic classification: Implications for tardive dyskinesia. *Modern Problems of Pharmacopsychiatry*, 21:49-64, 1983.

Jeste, D.V., and Wyatt, R.J. *Understanding and Treating Tardive Dyskinesia*. New York: The Guilford Press, 1982.

Kapit, R.M. Schizophrenia and tardive dyskinesia: Is schizophrenia also a "denervation hypersensitivity"? *Medical Hypotheses*, 3:207-210, 1978.

Kimura, D. Left hemisphere control of oral and brachial movements and their relationship to communication. *Philosophical Transactions of Royal Society. Series B: Biological Sciences*, B298:135-149, 1982.

Klawans, H.D., Jr. The pharmacology of tardive dyskinesia. *American Journal of Psychiatry*, 130:82-86, 1973.

Lapidot, M.B. Is there hemi-aging? In: Myslobodsky, M.S., ed. *Hemisyndromes: Psychobiology, Neurology, Psychiatry*. New York: Academic Press, 1983. pp. 193-212.

Marsden, C.D.; Tarsy, D.; and Baldessarini, R.J. Spontaneous and drug-induced movement disorders in psychotic patients. In: Benson, D.F., and Bloom, D., eds. *Psychiatric Aspects of Neurologic Disease*. New York: Grune & Stratton, 1973. pp. 219-266.

Meehl, P.E. Schizotaxia, schizotypy, and schizophrenia. *American Psychologist*, 17:827-838, 1962.

Mesulam, M.-M.; Waxman, S.G.; Geschwind, N.; and Sabia, T.D. Acute confusional states with right middle cerebral artery infarctions. *Journal of Neurology, Neurosurgery, and Psychiatry*, 39:84-89, 1976.

Mindham, R.H.S.; Gaind, R.; Antee, B.H.; and Rimmer, L. Comparison of amantadine, orphenadine, and placebo in the control of phenothiazine-induced parkinsonism. *Psychological Medicine*, 2:406-413, 1972.

Mintz, M., and Myslobodsky, M. Two types of hemisphere imbalance in hemi-parkinsonism coded by brain electrical activity and electrodermal activity. In: Myslobodsky, M.S., ed. *Hemisyndromes: Psychobiology, Neurology, Psychiatry*. New York: Academic Press, 1983. pp. 213-238.

Myslobodsky, M.S.; Holden, T.; and Sandler, R. Asymmetry of abnormal involuntary movements: A prevalence study. *Biological Psychiatry*, 19:623-628, 1984.

Myslobodsky, M.S.; Tomer, R.; Holden, T.; Kempler, S.; and Sigal, M. Cognitive impairment in patient with tardive dyskinesia. *Journal of*

*Nervous and Mental Disease*, 173:156–160, 1985.

Okinuora, M. Organic brain syndromes from a psychiatric point of view: Diagnostic and nosologic aspects. *Acta Neurologica Scandinavica*, 66 (Suppl. 92):47–57, 1982.

Owen, R.T. Dyskinesias. *Drugs of Today*, 15:65–80, 1979.

Oyewumi, L.K.; Lapierre, Y.D.; Gray, R.; Batth, S.; and Gelfand, R. Abnormal involuntary movements in patients on long-acting neuroleptics. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 7:719–723, 1983.

Rado, S., ed. *Psychoanalysis of Behavior: Collected Papers*. New York: Grune & Stratton, 1962.

Rajput, A.H.; Rozdilsky, B.; Hornykiewicz, O.; Shennak, K.; Lee, T.; and Seeman, P. Reversible drug-induced parkinsonism: Clinicopathophysiological study of two cases. *Archives of Neurology*, 39:644–647, 1982.

Reisberg, B.; Ferris, S.H.; and Gershon, S. An overview of pharmacological treatment of cognitive decline in the aged. *American Journal of Psychiatry*, 138:539–600, 1983.

Rosen, A.M.; Mukherjee, S.; Olarte, S.; Varia, V.; and Cardenas, C. Perception of tardive dyskinesia in outpatients receiving maintenance neuroleptics. *American Journal of Psychiatry*, 139:372–373, 1982.

Simpson, G.M. CNS effects of neuroleptic agents. *Psychiatric Annals*, 5:53–60, 1975.

Simpson, G.M., and Angus, J.W.S. A rating scale for extrapyramidal side effects. *Acta Psychiatrica Scandinavica*, Suppl. 212:11–19, 1970.

Simpson, G.M.; Lee, J.H.; Zoubok, B.; and Gardos, G. A rating scale for tardive dyskinesia. *Psychopharmacology*, 64:171–179, 1979.

Singh, M.M., and Kay, S.R. Dysphoric response to neuroleptic treatment in schizophrenia: Its relationship to autonomic arousal and prognosis. *Biological Psychiatry*, 14:277–294, 1979.

Smith, J.M.; Kucharski, L.T.; Oswald, W.T.; and Waterman, L.J. A systemic investigation of tardive dyskinesia in inpatients. *American Journal of Psychiatry*, 136:918–922, 1979.

Stevens, J.R., and Livermore, A. Telemetered EEG in schizophrenia: Spectral analysis during abnormal behaviour episodes. *Journal of Neurology, Neurosurgery, and Psychiatry*, 45:385–395, 1982.

Struve, F.A., and Wilner, W.E. Cognitive dysfunction and tardive dyskinesia. *British Journal of Psychiatry*, 143:597–600, 1983.

Vogel, H-P. Symptoms of depression in Parkinson's disease. *Pharmacopsychiatria*, 6:192–196, 1982.

Waziri, R. Lateralization of neuroleptic-induced dyskinesia indicates pharmacologic asymmetry of the brain. *Psychopharmacology*, 68:51–53, 1980.

Wegner, J.T.; Struve, F.A.; Kantor, J.S.; and Kane, J.M. Relationship between the B-mitten EEG pattern and tardive dyskinesia: A pilot control study. *Archives of General Psychiatry*, 36:599–603, 1979.

Weintraub, S.; Mesulam, M.-M.; and Kramer, L. Disturbances in prosody: A right hemisphere contribution to language. *Archives of Neurology*, 38:742–744, 1981.

Wilson, I.C.; Garbutt, J.C.; Lanier, C.F.; Moylan, J.; Nelson, W.; and Prange, A.J., Jr. Is there tardive dysmentia? *Schizophrenia Bulletin*, 9:187–192, 1983.

Winfield, D.L., and Sparer, P.J. The electroencephalogram in paralysis agitans. *Diseases of the Nervous System*, 15:114–117, 1954.

Wolf, M.E.; Ryan, J.; and Mosnaim, A.D. Organicity and tardive dyskinesia. *Psychosomatics*, 23:475–480, 1982.

Zoccolatti, P.; Scabini, D.; and Violani, C. Electrodermal responses in patients with unilateral brain damage. *Journal of Clinical Neuropsychology*, 4:143–150, 1982.

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