

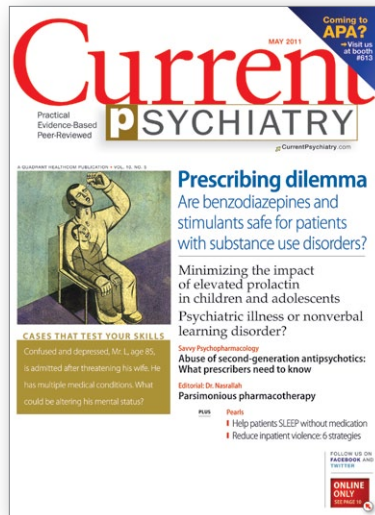
Differentiating NLD

“Does your patient have a psychiatric illness or nonverbal learning disorder?” (CURRENT PSYCHIATRY, May 2011, p. 17-35) reviews the differential diagnosis of “nonverbal learning disorder.”

Nonverbal learning disorder (NLD) has yet to be established as a distinct, valid neurodevelopmental syndrome. Many of the proffered diagnostic criteria, clinical presentations, and laboratory findings (especially results from psychological/neuropsychological and psychoeducational testing) overlap considerably with other neurodevelopmental syndromes characterized by social inadequacy and peculiarity, including Asperger’s disorder.

Therefore, rather than representing a discrete diagnostic category with overlapping features as suggested by this article, this putative syndrome probably is best conceptualized as falling within the milder end of a neurodevelopmental disorder spectrum characterized by deficits in social competence, judgment, and perception, accompanied by impaired daily functioning referable to these difficulties. The DSM-5 workgroup appears to recognize this problem of “splitting” vs “lumping.” Reports indicate that Asperger’s disorder may no longer be considered a “standalone” clinical syndrome and likely will be reconceptualized as a mild form of autism.

Many individuals have cognitive/neuropsychological and academic/learning difficulties compatible with nonverbal learning problems and do not exhibit the difficulties with social interactions the article cites. One way to advocate for NLD as a distinct clinical syndrome is to limit the diagnosis to a certain constellation of cognitive/



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neuropsychological and academic impairments that lead to educational, interpersonal, and/or vocational difficulties. Patients who also display clinically significant neurodevelopmentally based deficits in social competence/skills would fall outside this category and would be placed within the milder end of the autistic spectrum, or perhaps included within a broader “neuro-social disorder” spectrum. As the authors aptly point out, psychometric testing is important for diagnosis and psychoeducational planning for patients with the information processing difficulties and life struggles well described in this article.

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The authors respond

We thank Dr. Pollak for his comments. Although considerable overlap may exist among “neurodevelopmental syndromes characterized by social inadequacy and

peculiarity,” we sought to draw attention to the specific neurocognitive differences in NLD as compared with other disorders that, despite potentially sharing features with NLD, are distinct disorders.

We agree with Dr. Pollak’s suggestion that NLD someday might be classified within the DSM along a “neurodevelopmental disorder spectrum characterized by deficits in social competence, judgment, and perception accompanied by impaired everyday functioning referable to these difficulties.” This process will require careful characterization of individuals with NLD—as well as other individuals with difficulties in visual-spatial integration, attention, nonverbal memory, and expression and integration of emotion—to establish convergent and discriminant validity for NLD. Further, we believe that recognition of NLD as a distinct disorder will facilitate research and development of specific treatment interventions as compared with other conditions, despite potential syndromic or symptomatic overlap with NLD.

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Efficient pharmacotherapy

I agree with Dr. Nasrallah’s assertions that a single psychotropic can treat different disorders (“Parsimonious pharmacotherapy,” From the Editor, CURRENT PSYCHIATRY, May 2011, p. 12-

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16). During my consultation-liaison fellowship at MD Anderson Cancer Center, different forms of delirium were a daily occurrence in our hospitalized patients. A low dose of psychotropic—usually quetiapine—proved effective in controlling delirium; in acute and severe cases, low-dose IV haloperidol helped control agitation and psychosis.

Low-dose quetiapine also was beneficial in treating comorbidities such as increased anxiety, depression, pain, and insomnia, with minimal side effects. Because most of our patients suffered from several medical conditions and powerful, multiple-medication chemotherapies, in most cases psychotropic polypharmacy was contraindicated and avoided at all costs.

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Glutamatergic dysfunction

I read with interest the article by Drs. Kantrowitz and Javitt (“Glutamate: New hope for schizophrenia treatment,” *CURRENT PSYCHIATRY*, April 2011, p. 68-74) describing the glutamatergic model of schizophrenia. There is extensive evidence that in addition to neurocognitive deficits, schizophrenia is characterized by various neuromuscular abnormalities, including skeletal muscle fiber changes, alterations of alpha-motor neuron excitability, increased motor unit fiber densities, increased branching of terminal motor nerves, and elevated levels of muscular enzymes.^{1,2} These neuromuscular abnormalities also are found in healthy first-degree relatives of patients with schizophrenia.³ Although the precise cause of these neuromuscular abnormalities has not been elucidated, one possible explanation is that they may

be the result of neuronal injury mediated by excitatory amino acids.¹ For example, Stevens⁴ suggested that abnormal sprouting and reinnervation of neurons in schizophrenia might be caused by such injury.

Amyotrophic lateral sclerosis (ALS) is a progressive degenerative syndrome involving upper and lower alpha-motor neuron systems. A substantial body of evidence supports the hypothesis that glutamate-mediated excitotoxicity is responsible for the death of motor neurons in ALS.⁵ Evidence suggests that having schizophrenia may be associated with an increased risk of developing ALS, and this risk might be explained by the toxic effects of excitatory amino acids on neuronal function.^{1,6} Recently, Stommel et al⁷ hypothesized that treating schizophrenia could protect against development of ALS, which is of interest because antipsychotics may have direct and indirect effects on modulating glutamate receptor systems.⁸

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Treating delirium

Regarding “Atypical antipsychotics for delirium: A reasonable alternative to haloperidol?” (*CURRENT PSYCHIATRY*, January 2011, p. 37-46): Delirium usually is an acute encephalopathy with cerebral dysfunction caused by varying pathologies, most of which are extracranial. Examples include mental confusion induced by hypoxia or hypoglycemia. Primary treatment of delirium must be aimed at the specific cause.

Antipsychotic drugs are only a symptomatic intervention for delirium. They can and do provide behavioral control; however, these medications may worsen cases of alcohol or sedative withdrawal, ictal-related problems, neuroleptic malignant syndrome, etc. An antipsychotic may complicate other conditions, thus creating additional clinical difficulties for some patients. Recommending antipsychotics as a treatment focuses on symptomatic aspects; however, the critical mandate is to diagnose and specifically manage the etiology. Once the cause is corrected, the delirium usually resolves.

While treating the primary pathology, if behavioral issues still urgently require immediate control, a benzodiazepine is safer than an antipsychotic. Both medications provide symptomatic control, but a benzodiazepine is less likely to add new clinical problems. The only major precaution with a benzodiazepine is to avoid overprescribing. It is simply safer to rely on benzodiazepines for short-term behavioral management.

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