patient, a 30-year-old man, was taking lithium carbonate, 900 mg/day, with an average serum lithium level of 0.7 mmol/L. The patient's lesion resolved in 15 days after he discontinued the lithium against medical advice. Patki noted that geographic tongue is a minor side effect of lithium therapy and does not warrant withdrawal unless generalized pustular psoriasis occurs. A second case reported by Nathan (1995) occurred in a 56-year-old woman with mood disorder believed to be secondary to Graves disease; she developed mucosal ulcerations in the mouth, geographic tongue, and vaginal ulcerations at a dose of slow-release lithium carbonate 300 mg p.o. t.i.d., with a serum level of 0.9 mEq/L. After the patient switched from lithium to sodium valproate, the lesions resolved.

The incidence of geographic tongue as a side effect of lithium treatment appears to be relatively rare, and it was previously reported only in adults. Geographic tongue is usually asymptomatic, with occasional complaints of burning. Lesions heal without scar formation. Mouth rinses or topical creams may be used for symptomatic relief (Demis, 1998, p. 5).

Child psychiatrists should be sensitive to patients' complaints of mucosal membrane or tongue pain or ulcerations and should consult a dermatologist for assistance in diagnosis and management.

Barbara L. Gracious, M.D.
University Hospitals of Cleveland
Case Western Reserve University
Cleveland
Maria Llana, Pharm.D., M.A.
Division of Child and Adolescent Mental Health
Scott & White Clinic
Texas A&M University
Temple, TX
David D. Barton, M.D.
Department of Medicine, Dermatology Division
Scott & White Clinic
Texas A&M University
Temple, TX

Demis DJ, ed (1998), *Clinical Dermatology*, Vol 4, Unit 28–1: *Oral Medicine.* Philadelphia and New York: Lippincott-Raven

Litt JZ (1998), *Drug Eruption Reference Manual, DERM.* New York: Parthenon Publishing Group

Nathan KI (1995), Development of mucosal ulcerations with lithium carbonate therapy. *Am J Psychiatry* 152:956–957

Patki AH (1992), Geographic tongue developing in a patient on lithium carbonate therapy (letter). *Int J Dermatol* 31:368–369

CRIMINAL RECIDIVISM AS A NEUROBEHAVIORAL SYNDROME

To the Editor:

We wish to propose the utility of a previously unrecognized clinical syndrome which, arising from abnormal function of the reward system, progresses from attachment disorders to the early use of alcohol and drugs with subsequent development of Cloninger type 2 alcoholism (Cloninger, 1987) and thence to the accumulation of brain damage through both mechanical and chemical means. This developmental path commonly ends in chronic criminal recidivism with its attendant and enormous financial, social, and personal costs.

We suggest that the underlying neurological abnormalities that drive this series of developmental deviations include an abnormally high reward threshold, as has been documented in boys with attention-deficit hyperactivity disorder (ADHD) (Ornitz et al., 1997). A neurophysiological mechanism which could create a high reward threshold has been described: neurons of the nucleus accumbens, which is a critical component of the reward system and is critically involved in addictive behavior (Koob and Nestler, 1997), may be either hypo- or hyperpolarized (Heimer et al., 1997). The hypopolarized neurons, which are close to their firing state at rest, must be firing in bursts at frequent intervals, thereby "drip-irrigating" multiple brain areas which, in aggregate, are critical to pleasure, well-being, and satisfaction. The hyperpolarized neurons, on the other hand, obviously are "special occasion" neurons which fire only when vigorously stimulated.

It follows that genetic variation would create subpopulations of individuals with varying distributions of these neurons and that some individuals, because of an excessive population of hyperpolarized nucleus accumbens neurons, will be unable to harvest an adequate firing rate from ordinarily available inputs. Correspondingly, the individual would find life uninteresting without highly intense stimulation. Every developmental level must suffer distortion, beginning with basic trust and attachment, which must be impaired as the caretaker–infant system struggles to compensate for the infant's damaged reinforcement sensitivity. The child's efforts to reduce the resulting pervasive distress and lack of satisfaction, which must be continually exacerbated by the high reward threshold, logically lead to behaviors which present clinically as Cloninger type 2 alcoholism: the early onset of alcohol abuse, reward independence, and high-risk, exciting behaviors such as fighting. The problematic behaviors fall into 2 categories: (1) the use of alcohol and other sedatives in attempts to calm the distress which inevitably arises from a lack of pleasure, satisfaction, and ordinary well-being; and (2) high-risk behaviors and use of stimulant drugs in attempts to stimulate the reward system.

The next step in this sequence is the accumulation of brain damage through both chemical and mechanical means. The resulting impulsivity arising from forebrain and temporal lobe damage and the yet-further increases in the reward threshold resulting from both cortical damage (Heimer et al., 1997) and exposure to drugs of abuse (Koob and Nestler, 1997), create a positive-feedback loop which creates a catastrophic, rapidly

accelerating "tailspin" downward into a disastrous combination of drug and alcohol dependence and high-risk behaviors with the simultaneous decline in overall coping ability.

The final common pathway of this entire process is chronic criminal recidivism as the syndrome runs its full course to a level which requires highly structured institutional management.

The remarkably consistent emergence of this pattern in individuals of widely divergent socioeconomic and social backgrounds and recent reports of neurophysiological mechanisms which might reasonably be expected to account for this behavioral sequence embolden us to propose that this developmental sequence and its underlying neurological basis constitute a clinical syndrome.

These children present clinically with ADHD, intermittent explosive disorder, mood disorders, and other problems which are the daily fare of child psychiatry. Conceptualization of this sequence as the relatively predictable unfolding of a neurological disorder may reduce such maladaptive responses as guilt, rage, and rejection and may mobilize early and life-saving interventions before brain damage worsens prognosis.

Alfred P. French, M.D.
Roseville, CA
Daniel G. Amen, M.D.
Director, Amen Clinic for Behavioral Medicine
Fairfield, CA

Cloninger CR (1987), Neurogenetic adaptive mechanisms in alcoholism. *Science* 236:410–416

Heimer L, Alheid G, de Olmos J et al. (1997), The acumbens beyond the core-shell dichotomy. In: *The Neuropsychiatry of Limbic and Subcortical Disorders,* Salloway S, Malloy P, Cummings J, eds. Washington, DC: American Psychiatric Press, PP 43–70

Koob G, Nestler EJ (1997), The neurobiology of drug addiction. In: *The Neuropsychiatry of Limbic and Subcortical Disorders,* Salloway S, Malloy P, Cummings J, eds. Washington, DC: American Psychiatric Press, pp 179–194

Ornitz EM, Gabikian P, Russell AT, Guthrie D, Hirano C, Gehricke JG (1997), Affective valence and arousal in ADHD and normal boys. *J Am Acad Child Adolesc Psychiatry* 36:1698–1705

CRITERIA FOR ASPERGER'S DISORDER

To the Editor:

There is considerable criticism of the *DSM-IV* (American Psychiatric Association, 1994) definition of Asperger's disorder, in particular the criterion that there should be no clinically significant general delay in language. It is commonly suggested that this criterion be removed from future revisions of *DSM-IV*. Unfortunately, this suggestion would create as many problems as it would solve, as then persons with Asperger's disorder who have no clinically significant general delay in language would be excluded.

The following are 2 suggestions for a future revision of *DSM-IV* in relation to Asperger's disorder.

(a) Section D. Communication. One of the following:
  (1) no clinically significant general delay in language
  (2) delay in the development of spoken language
  (3) impairment in the ability to initiate or sustain conversation with others
  (4) stereotyped or repetitive use of language or idiosyncratic language

(b) An alternative would be to remove Section D entirely from the Asperger's disorder definition and to have an additional coding for:
  A. No clinically significant general delay in language development.
  B. Impairment in speech and language.

At least for research purposes, this would allow researchers who are interested in Asperger's disorder with or without clinically significant general delay in language to study these groups separately.

Michael Fitzgerald, M.D.
Henry Marsh Professor Child & Adolescent Psychiatry T.C.D.
Dublin, Ireland

American Psychiatric Association (1994), *Diagnostic and Statistical Manual of Mental Disorders, 4th ed (DSM-IV).* Washington, DC: American Psychiatric Association

---

The Letters column is a corner of the *Journal* that encourages opinion, controversy, and preliminary ideas. We especially invite reader comments on the articles we publish as well as issues or interests of concern to child and adolescent psychiatry. The Editor reserves the right to solicit responses and publish replies. All statements expressed in this column are those of the authors and do not reflect opinions of the *Journal.* Letters should not exceed 750 words, including a maximum of 5 references. **They must be signed, typed double-spaced, and submitted in duplicate.** All letters are subject to editing and shortening. They will be considered for publication but may not necessarily be published nor will their receipt be acknowledged. Please direct your letters to Mina K. Dulcan, M.D., Editor, Journal of the AACAP Editorial Office, Children's Memorial Hospital, 2300 Children's Plaza #156, Chicago, IL 60614-3394.