

responded favorably to lorazepam, 2 mg/day. For the past year, he has been receiving treatment with clozapine and lithium and remains in full remission of catatonic, mood, and psychotic symptoms, with dramatic improvement in social, interpersonal, and educational functioning.

Mr. B, an 18-year-old Caucasian man with a history of cannabis abuse and declining social, vocational, and interpersonal involvement (but no previous diagnoses of mood or psychotic disorder), was admitted because he was talking to himself, had lack of motivation, and was laughing inappropriately. He exhibited profound mutism, intermittent excitement, posturing, staring, mannerisms, stereotypy, perseveration, autonomic abnormality (elevated blood pressure), automatic obedience, and impulsivity. His Bush-Francis Catatonia Rating Scale score was 24. His catatonic signs responded well to lorazepam, 3 mg/day. Manic symptoms subsequently emerged, and Mr. B was diagnosed and treated for bipolar disorder.

Carl, a 17-year-old Caucasian youth who was previously diagnosed with schizoaffective disorder, was admitted because of declining self-care, response to internal stimuli, and bizarre delusions. He displayed facial grimacing that resolved completely when risperidone, 5 mg/day, was discontinued. His Bush-Francis Catatonia Rating Scale score was 18, and his catatonic signs included immobility, mutism, excitement, posturing, staring, mannerisms, echolalia, stereotypy, negativism, gegenhalten, ambitendency, impulsivity, and combativeness. These signs resolved with lorazepam treatment, 3 mg/day.

All youths had negative serum toxicology screens upon admission to the facility and, in the case of Mr. B, for 6 months before admission, as verified through court-ordered monitoring. The youths received medical and neurological evaluations, including hematological, metabolic, toxicological, and CSF analysis, EEGs, and neuroimaging. All results were normal or lacked positive findings. Prenatal and developmental histories were unremarkable, although Abe and Carl had extensive family histories of mental illness.

There have been several case series and reports of catatonia occurring in the child and adolescent population (2). Although substantial psychiatric morbidity has been identified among youths in the juvenile justice system (3), I am unaware of previous case reports of catatonia occurring among youths detained within the juvenile justice system. Presumably, the etiology and risk factors leading to catatonia in adolescents and young adults in juvenile justice and community settings is similar, regardless of criminal history. However, the identification of this syndrome in male juvenile offenders is especially important, given the paucity of resources for adolescents and the increasing recognition of the prevalence and severity of mental illnesses among juvenile offenders (4). Incarcerated juveniles may exhibit unusual behavioral phenomena, making detection of psychiatric disorders in need of treatment extremely difficult (5).

Considerable functional improvement was evident in all three cases after treatment of catatonia, similar to documented case reports. Also, more youths are entering the criminal justice system than ever before. With more diverse clinical treatment settings and declining resources, greater awareness of the catatonia syndrome, with its well-defined features and response to treatment (6), may aid in its recognition and management.

## References

1. Standardized instruments, in *Catatonia: From Psychopathology to Neurobiology*. Edited by Carnoff SN, Mann SC, Francis A, Fricchione GL. Arlington, Va, American Psychiatric Press, 2004
2. Takaoka K, Takata T: Catatonia in childhood and adolescence. *Psychiatry Clin Neurosci* 2003; 57:129–137
3. Teplin LA, Abram KM, McClelland GM, Dulcan MK, Mericle AA: Psychiatric disorders in youth in juvenile detention. *Arch Gen Psychiatry* 2002; 59:1133–1143
4. Coccozza JJ, Skowrya KR: Youth with mental health disorders: issues and emerging responses. *Juvenile Justice* 2000; 7:3–13
5. Nurcombe B, Mitchell W, Begtrup R, Tramontana M, LaBarbera J, Pruitt J: Dissociative hallucinosis and allied conditions, in *Psychoses and Pervasive Developmental Disorders in Childhood and Adolescence*. Edited by Volkmer F. Washington, DC, American Psychiatric Press, 1996, pp 107–128
6. Taylor MA, Fink M: Catatonia in psychiatric classification: a home of its own. *Am J Psychiatry* 2003; 160:1233–1241

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## Overlap Between Alexithymia and Asperger's Syndrome

TO THE EDITOR: It seems to us that there is a significant overlap between alexithymia and Asperger's syndrome. The term "alexithymia" was coined by Sifneos in 1972. It is derived from the Greek, with *alexi* meaning "no words" and *thymia* meaning "mood or emotion." Patients with alexithymia have great difficulty or are unable to describe their feelings and can have problems making sophisticated differentiation of one feeling from another. Their communicative style shows markedly reduced or absent symbolic thinking (1). As Warnes (2) pointed out, they have "a paucity of fantasies" and "lack the capacity for introspection." They are preoccupied with the "minute detail of external events...[and] are unable to make connections between events, affective arousal and somatic response." Nonverbally, they are "stiff and wooden." They are "mechanical in their object relations." Alexithymic individuals give flat, shallow descriptions of others that lack "psychological counters" (2).

All of these features also fit descriptions of Asperger's syndrome (3), in which the main difficulties are understanding one's own and others' emotions, having problems expressing oneself with nonverbal behavior and in reading that of others, and having a propensity for hypochondriacal features. They also have difficulty with the "theory of mind" and in predicting the cognitions of others. Their imagination is limited. They tend to have a preoccupation with factual information and are strong in areas such as mathematics, engineering, and computers but can have significant problems with interpersonal relationships.

It appears to us that from a clinical perspective a diagnosis of Asperger's syndrome should be considered in patients with alexithymia.

## References

1. Taylor GJ: Alexithymia: concept, measurement, and implications for treatment. *Am J Psychiatry* 1984; 141:725–732
2. Warnes H: Alexithymia—clinical and therapeutic aspects. *Psychother Psychosom* 1986; 46:96–104

3. Asperger H: Die "Autistischen Psychopathen" im Kindesalter (1944), in *Autism and Asperger Syndrome*. Edited by Frith U. Cambridge, UK, Cambridge University Press, 1991, pp 37–92

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## Franz Alexander

TO THE EDITOR: I would like to comment on the article by Judd Marmor, M.D., that appeared recently in *Images in Psychiatry* (1). Dr. Marmor wrote that Franz Alexander "was invited in 1930 by Robert Hutchins, then President of the University of Chicago, to become its Visiting Professor of Psychoanalysis—the first University Chair of Psychoanalysis in history."

Dr. Marmor's statement is true from a practical point of view since Alexander was the first *functioning* head or chair of psychoanalysis in history. However, the historical truth is that the first psychoanalyst who was appointed to be a professor of psychoanalysis was Sándor Ferenczi (2). He received this title at the University of Budapest in 1919 by the short-lived communist regime. After the change in regime, Ferenczi's appointment as the newly founded chair was not confirmed, so he lost the appointment before he was able to begin to function as a university professor of psychoanalysis.

## References

1. Marmor J: Franz Alexander, 1891–1964 (image, psych). *Am J Psychiatry* 2002; 159:1305
2. Stanton M: Sándor Ferenczi: Reconsidering Active Intervention. London, Free Association Books, 1990

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## Anorexia Nervosa and Gastrointestinal Tumors

TO THE EDITOR: As pointed out by Katherine A. Halmi, M.D., and Gladys Frankel, Ph.D., (1), the process of differential diagnosis between anorexia nervosa and anorexia due to gastrointestinal stromal tumors is sometimes problematic. The authors emphasized the importance of making a diagnosis based on positive criteria.

Nevertheless, even if the diagnosis of anorexia nervosa is evident in the case reported in their article, because the development of progressive malnutrition or cachexia is frequent in patients with gastrointestinal cancer, it still raises certain questions.

Cachexia syndrome is characterized by an involuntary weight loss of more than 5% of premorbid weight occurring within 6 months and often associated with anorexia and fatigue. Moreover, anorexia has been reported in patients with gastrointestinal stromal tumors, but unlike anorexia nervosa, it was not associated with voluntary weight loss and bingeing/purging behavior but was frequently associated with the presence of fever.

In this case, the negative criteria should also be considered, notably the absence of fever, fatigue, cachexia syndrome, and nausea and abdominal pain, which may have induced intentional anorectic behavior in the early onset of the disease.

Anorexia nervosa and cachexia are two distinct syndromes that may have synergistic effects in patients. Moreover, the

occurrence of metastasis in the case report might have increased weight loss or, as pointed out by the authors, it might have been preceded by weight loss, which may lead to a perpetual cycle of maintaining weight loss and malnutrition. As the authors stated, when the tumor became a large mass, it could have created an early experience of satiety, and a highly restrictive diet might have been reinforced by undetected gastrointestinal stromal tumors.

Hence, this case report is an interesting and exceptional case of anorexia due to comorbid diagnoses. From a psychoneuroendocrinological point of view, this case deserves further attention.

Several studies in anorectic patients have found increased levels of pro-inflammatory cytokines, such as interleukin (IL) IL-1, IL-6, and tumor necrosis factor alpha (known for their anorexigenic effects), indicating autoimmune activation (2). Pathophysiological parallels have been drawn between the role of cytokines in cancerous cachexia and their putative involvement in the undernourished states observed in anorexia nervosa (3, 4). Tumor necrosis factor alpha, IL-1, IL-6, and interferon  $\gamma$  have been proposed as mediators of the cachectic process. It has been shown that the levels of these cytokines correlate with the progression of the tumors (5).

Therefore, it is not unreasonable to consider that the assay of pro-inflammatory cytokines, such as tumor necrosis factor alpha, in this case might have been a good marker of the course of the disease and might have helped to disentangle the evolution and the contribution of the two disorders to weight loss when we considered serum levels of this cytokine.

## References

1. Frankel GJ, Halmi KA: An adolescent with anorexia nervosa and gastrointestinal stromal tumors (clin case conf). *Am J Psychiatry* 2003; 160:1056–1059
2. Corcos M, Guilbaud O, Paterniti S, Moussa M, Chambry J, Chaouat G, Consoli SM, Jeammet P: Involvement of cytokines in eating disorders: a critical review of the human literature. *Psychoneuroendocrinology* 2003; 28:229–249
3. Holden RJ, Pakula IS: The role of tumor necrosis factor-alpha in the pathogenesis of anorexia nervosa and bulimia nervosa, cancer cachexia and obesity. *Med Hypotheses* 1996; 47:423–438
4. Plata-Salaman CR: Cytokines and anorexia nervosa: a brief overview. *Semin Oncol* 1998; 25(suppl 1):64–72
5. Matthys P, Biliau A: Cytokines and cachexia. *Nutrition* 1997; 13: 763–770

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## Drs. Halmi and Frankel Reply

TO THE EDITOR: We agree with Dr. Guilbaud and his associates that measurement of pro-inflammatory cytokines may be helpful in following the progression of gastrointestinal stromal tumors. In fact, most patients with anorexia nervosa do not have elevated cytokine levels; thus, an increase in these