

induced aCL remains unclear. The induction of aCL requires β -2-glycoprotein-1 as a cofactor for binding to cardiolipin.⁶ It is assumed that neuroleptics can increase phospholipid expression and activation on the cell membrane, inducing aCL binding to cardiolipin epitopes without involving β 2-glycoprotein-1. This causes the induction of mainly IgM-aCL rather than IgG-aCL (IgG-aCL is the main isotype related to thrombosis). This might explain the lack of vascular events in these patients.

We earlier reported the presence of LA activity and IgG-aCL in a group of patients with psychosis who were not taking medication and had no clinical evidence of vasculopathy.¹ These findings, together with our present study, raise the possibility that these autoantibodies are implicated in the etiology or course of psychosis. Sirota et al⁷ reported the presence of different types of autoantibodies, including aCL, in patients with schizophrenia and their healthy first-degree relatives. Firer et al⁸ found aCL antibodies in both patients with schizophrenia and their relatives. Roy Chengappa et al⁹

propose that raised titres of IgG-aCL may be a marker for autoimmune reactivity or may play a role in schizophrenia. To support such a possibility, both the patients and controls were tested for the presence of anti-nuclear antibodies. The negative result in both groups does not support this notion; however, it can not be excluded, since the sample was relatively small.

This study re-established the association between long-term treatment with neuroleptic medication and LA and elevated aCL. Further studies are needed to clarify the role of antiphospholipid antibodies in these patients.

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Understanding autism: does anyone read new medical journals?

Dear Sir:

I read Dr. Peter E. Tanguay's editorial¹ with mounting interest after he started by citing Barr's 1898 case of autism,² since this is my first indication that anyone has read my 1982 letter drawing attention to this case.³ Tanguay stated, "In this issue of the Journal (see page 103), Drs. Trottier, Srivastava and Walker present a remarkable review of this current literature. Their report is lively, well

organized and very comprehensive." However, I was disappointed to find that this review article, with 124 references, made no mention of possible peripheral auditory defects or ear disease in autism, despite noting the relevance of sensory pathway integrity and devoting a column to auditory impairment.

For a quarter of a century I have been proposing that autism is an unusual variant of peripheral deafness.³⁻²⁴ Surely it is time that some-

one somewhere took this theory seriously and discussed it. If false, it should be trashed, if only to stop further valuable journal space being devoted to a nonviable theory. It is simple to the point of naïveté, so it should be easily refutable if it is incorrect. For example, all that is needed is to find some risk maker or risk marker for autism that is not also associated with middle- or inner-ear deafness.¹⁷ I have looked quite hard and still not found one,

although I cannot find auditory data for some conditions, such as tuberous sclerosis.

In the review article, one sentence alone excludes primary brain damage in autism: "The intellectual competency of children with autism ranges from profound mental retardation to superior intelligence." What other developmental condition, with proven brain lesions, has this same IQ distribution and pattern of verbal and nonverbal IQ?

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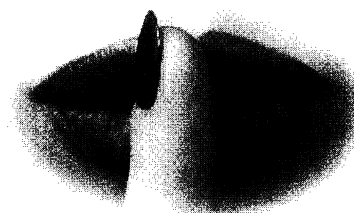
[The author replies:]

Thank you for your 1982 letter calling attention to Barr's case report. I did not enter the letter into my reference collection at the time; hence, I was unable to credit you for this help. Does auditory dysfunction lead to autism? Auditory event-related potentials have not reliably identified brainstem abnormalities in autistic children. Solid, replicable and convincing evidence of central or peripheral auditory dysfunction will be needed before we can attribute autism to this cause.

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An Unspoken Side Effect



Its rate may be higher than you think.

Recent reports suggest that **40-60%** of patients may experience sexual dysfunction with some classes of antidepressants (SSRIs), although rates are not reflected in product monographs.

(New 1999 C.A.N.M.A.T. Guidelines*)

* Guidelines for the Diagnosis and Pharmacological Treatment of Depression. Canadian Network for Mood and Anxiety Treatment (C.A.N.M.A.T.) Depression Working Group 1st Edition 1999.

