Clomipramine: positive results

A successful test of the drug clomipramine for autism adds evidence to the "serotonin theory" of autism.

Charles Gordon et al. conducted a double-blind crossover study of seven autistic children, six to 18 years of age, under three conditions:

—Two weeks on a placebo.

—Five weeks of treatment with clomipramine, a drug used to treat depression and obsessive behaviors. Clomipramine blocks the reuptake by brain cells of the brain messenger chemical serotonin, a substance which appears to be abnormally high in about 30% of autistic children.

—Five weeks of treatment with desipramine, an antidepressant which does not directly alter serotonin levels.

The researchers report that "clomipramine was superior to desipramine and placebo" in reducing the autistic children's repetitive and compulsive behaviors. The children's core autistic symptoms and anger levels lessened, and the researchers noted a trend toward less deviant speech. The parents of all seven subjects chose to have their children continue taking clomipramine after the study ended.

Desipramine — the drug which did not affect serotonin levels — was more effective than the placebo in controlling hyperactivity, but did not affect the children's obsessive-compulsive behaviors.

Side effects of clomipramine treatment were mild, and included disturbed sleep, dry mouth, constipation, and the development of a mild tremor in one subject. Two children were removed from the desipramine trial after becoming extremely irritable and having temper outbursts.

Autism, OCD similarities

Gordon et al. say the similar symptoms of autism and obsessive-compulsive disorder, and the reduction of symptoms in both conditions when serotonin reuptake-blocking drugs such as clomipramine are administered, "suggest similarities in underlying pathophysiology." It is possible, they say, that "central serotonergic dysfunction might be related to impulsive, ritualistic behavior in general, independent of diagnostic categories."

They note, however, that the mechanism by which clomipramine reduces autistic symptoms is unknown, and add that the drug fenfluramine—which lowers serotonin levels—has shown equivocal results in most trials on autistic children.

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"Differential response of seven subjects with autistic disorder to clomipramine and desipramine," Charles T. Gordon, Judith L. Rapoport, Susan D. Hamburger, Rosanne C. State, and Glenn B. Mannheim; *American Journal of Psychiatry*, 1992, 149:363-366. Address: Charles Gordon, Child Psychiatry Branch, NIMH, Bldg. 10, Room 6N240, 9000 Rockville Pike, Bethesda, MD 20892.

"Autism" develops in 31-year-old

Swedish researcher I. Carina Gillberg recently reported the case history of a 31-year-old man who developed autism after contracting herpes encephalitis (inflammation of the brain following herpes infection). While cases of a 13-year-old and an 11-year-old developing autism after encephalitis have previously been reported, Gillberg believes this "possibly is the first adult case in the literature."

Before contracting herpes encephalitis, Gillberg notes, the man "had never been psychiatrically ill, deviant, or even odd; and in every respect was considered a healthy man." Following the infection—which caused temporal lobe damage and seizures—the man was hypersensitive to noise, aloof, and aggressive; avoided social interaction and eye contact; spoke only in echolalic words and phrases; and developed a number of stereotyped behaviors and rituals. Except for age of onset, Gillberg says, the man clearly met the criteria for autistic disorder. He is now 48, and continues to exhibit all of the symptoms of autism.

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"Autistic syndrome with onset at age 31 years: herpes encephalitis as a possible model for childhood autism," I. Carina Gillberg; *Developmental Medicine and Child Neurology*, 1991, 33, pp. 912-929. Address: I. Carina Gillberg, Department of Pediatrics and Child Psychiatry, Child Neuropsychiatry Center, University of Goteborg, Box 17113, S-402 61 Goteborg, Sweden.

Treating tardive dyskinesia with vitamin E: more positive results

Autistic persons are often significantly impaired by tardive dyskinesia (TD), the chronic involuntary muscle movements frequently caused by prolonged use of neuroleptic drugs such as Haldol. ARRI reported in 1991 (5/1) that vitamin E may be an effective treatment for TD. At that time, researcher A.M. Elkashef and colleagues noted that five of eight patients treated with vitamin E showed improvements of 30% or more on the Abnormal Involuntary Movement Scale. (The patients had shown symptoms of TD for an average of 3.8 years.)

Now a new study by New York researcher Lenard A. Adler offers more evidence that vitamin E is an effective weapon against TD. Adler found that symptoms of tardive dyskinesia decreased significantly in nine of 16 psychiatric patients he treated with large daily doses of vitamin E. Only one patient in a matched placebo group showed improvement.

Adler et al. note that younger patients showed more improvement than older patients: four of five patients under 58 showed significant improvement, compared with only five of 11 patients over that age.

Earlier studies in the late 1980s also provided strong evidence that vitamin E can reduce symptoms of tardive dyskinesia, and may even prevent the disorder from occurring. Jean Lud Cadet and James Lohi reported in 1989 that almost half of 15 TD sufferers treated vith vitamin E improved, while David Hawkins found that only .05% of 61,000 patients given neuroleptic drugs developed TD if also given high doses of vitamins B3, B6, C and E.

While the reasons for the vitamin's effectiveness in reversing tardive dyskinesia are unknown, Sally Szymanski suggests that it protects the brain from damage caused by toxic molecules known as "free radicals" produced when neuroleptic drugs alter the metabolism of natural brain substances. C.P. LeBel agrees, noting that brain tissue is especially sensitive to a deficiency of vitamin E because the membranes of brain cells contain large amounts of polyunsaturated fats, which are prone to damage by free radicals.

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"Vitamin E may ease movement disorder," Bruce Bower, *Science News*, May 23, 1992, page 351.

—and—

"Vitamin E increasingly recognized for preventing brain damage and cancer," *The Nutrition Reporter*, Vol. 3, No. 2, 1992, pp. 1-4.

Recently published . . .

THERE'S A BOY IN HERE, Sean and Judy Barron, Simon & Schuster, 1992, 246 pages.

Fascinating book about a young man who overcame autism. Sean and his mother describe how his obsessions ruled his life, and how his mother's determination helped him overcome his disability.

NEUROBIOLOGY OF INFANTILE AUTISM, H. Naruse and E. M. Ornitz, eds., Elsevier Science Publ. Co., P.O. Box 882, Madison Square Garden Station, New York City, NY 10159, 416 pages, $143.00.

Proceedings of an international symposium in Tokyo in November 1990. Thirty-nine papers on a very wide range of topics on the biology of autism, including histological and imaging studies, biochemistry, pharmacology and brain models. Much important, up-to-date information for professionals.

SILENT WORDS/FOREVER FRIENDS, Margaret and David Eastham, Oliver-Pate Publishers, P.O. Box 4017, Station E, Ottowa, Ontario, K1S 5BI, Canada, 1992, $24.00 Canadian, $20.50 U.S.

David, a non-speaking but gifted autistic poet, died of drowning at age 24. Margaret Eastham's intensely interesting biography of her son (*Silent Words*) is complemented here with a collection of his poems (*Forever Friends*). Much information in this book on what is now called Facilitated Communication.