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Dietary Management of Nystagmus

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Summary

Two case reports illustrate the therapeutic response of congenital nystagmus to a diet eliminating synthetic food colors, synthetic food flavors, the antioxidant preservatives butylated hydroxytoluene (BHT) and butylated hydroxyanisole (BHA), and a small group of foods thought to contain a natural salicylate radical.

A brief discussion of the hyperkinetic syndrome is offered with the proposal that a variety of neurologic and neuromuscular disturbances (grand mal, petit mal, psychomotor seizures; La Tourette syndrome; autism; retardation; the behavioral component of Down's syndrome; and oculomotor disturbances) may be induced by identical chemicals, depending upon the individual's genetic profile and the interaction with other environmental factors. It is perhaps the failure to integrate all the signs presented by the various clinical patterns with hyperkinesis or Minimal Brain Dysfunction (MBD) under a single heading that eye muscle involvement manifested as either nystagmus or strabismus has not been emphasized as part of the hyperkinetic syndrome.

Observations linking behavioral disturbances and learning disabilities to the ingestion of synthetic food colors and flavors, and to a small group of foods thought to contain a natural salicylate radical, were reported in 1973 [1] and then again in 1974 [1]. Following the initial announcement, worldwide interest developed in this relationship. Thousands of children suffering from behavioral disturbances labelled as Hyperkinesis or Minimal Brain Dysfunction (MBD) with and without learning disabilities reportedly experienced favorable responses to dietary intervention [2]. Many of these children who had

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failed to respond clinically to various behavior-modifying modalities as well as counselling, psychotherapy and medications (Ritalin, amphetamines, Stelazine, Mellaril, Elavil, Cylert, Tofranil, etc.), showed an early and at times dramatic improvement on dietary management.

Initially, the professional acceptance of the hypothesis was limited. The diet was criticized as being non-nutritious because of vitamin C deficiency [3]. However, diet analyses refuted this argument [4]. The validity of the basic hypothesis was then challenged with the contention that the favorable response was induced by non-specific factors [3], *e.g.* placebo effects, including increased family dynamics, Hawthorne[1] effects, etc. Over the last five years these criticisms have also been answered by several controlled blind clinical studies [5] and, more recently, by observations on rats [6]. During this same period the base of our clinical observations has been broadened to include a variety of neurologic and neuromuscular disorders [6].

The clinical patterns that particular individuals exhibit after consuming the food additives described above differ; such differences may reflect their genetic profiles or interactions with other environmental factors. As a result a single compound apparently can produce in addition to hyperkinesis or MBD clinical patterns resembling those shown in a variety of neurologic disorders, including petit mal and grand mal epilepsy; psychomotor seizures; La Tourette syndrome; retardation; autism; the behavioral component of Down's syndrome; and oculomotor disturbances, *e.g.* nystagmus.

The following case reports illustrate therapeutic responses of congenital nystagmus to dietary intervention in two children.

Case 1[2]. A male child, the product of a full-term normal pregnancy and uneventful delivery was born on October 26, 1965 with a birth weight of 6 lb. 10 oz. Growth and development during infancy were normal.

About January 1966, at 3 months of age, the mother noted the eyes were turning from time to time.

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[1] The Hawthorne or halo effect are terms applied to the bias of an interviewer or rater attributed to the influence of knowledge concerning the previous performance of the subject, or to the bias induced by favorable or unfavorable prejudice of the observer or interviewer.—References: *Fantino, E., Reynolds, G.:* Contemporary Psychology, p. 479. San Francisco: W. H. Freeman. 1975. *Bannatyne, A.:* Language, Reading and Learning Disabilities, pp. 340, 456. Springfield, Ill.: Charles C Thomas. 1971.

[2] Historical data furnished by H. Jerome Crampton, M.D., Lawrence, Massachusetts.

August 1966, at 8 months of age, the pediatrician referred the child to an ophthalmologist because of "drifting eyes".

The ophthalmologist's initial examination reported, "Coarse horizontal and rotary nystagmus of both eyes with intermittent exotropia, alternating in type. There was a marked dominance of the left eye. Fundus was normal with a good pink nerve head. Refractive error was minimal on the hyperopic side. The diagnosis is congenital nystagmus of unknown etiology; exotropia with left eye dominance."

The child was placed on alternate use of atropine for about 3 months, following which examination revealed the eyes to be alternating freely. Atropine was discontinued.

The child was referred to the Massachusetts Eye and Ear Infirmary in Boston, where the previous findings were confirmed, corroborating a diagnosis of congenital nystagmus of unknown etiology.

The child was examined at annual intervals until school age, when more definitive measurements of vision were possible.

In 1971, at age six, the vision of either eye was 20/400 separately and 20/300 together; near vision was 20/100. There was no refractive error. Books with large type were ordered for him at school.

The child's behavior during this period was marked by intermittent tantrums. The child would fall to the floor, with head knocking, crying and destructiveness of any object within reach. At school the child was disturbed by the taunting of his peers who called him "typewriter eyes".

Ritalin, 2.5 mg b.i.d., offered only intermittently to permit the child to attend school, was discontinued after about four years.

Ophthalmological examinations were conducted in 1972, 1973 and 1975, with no change in the nystagmus.

In January 1976 the mother started the elimination diet (see Table 1).

In October 1976 the child was re-evaluated on a routine basis, at which time the mother stated he was backing away while watching television, and the movement of his eyes seemed quite less active. At this time the mother reported to the ophthalmologist that the child had been less hyperactive since starting on dietary control. The mother also reported that the child was shooting clay pigeons with his father and improving each month.

Examination in October 1976 revealed the vision of either eye was corrected to 20/80 at distance, separately or together, and 20/25 near at 4 inches. Nystagmus was much less marked with both eyes open.

In June 1977 while still on dietary control his vision was 20/80 in either eye separately or 20/50 together, and near vision was 20/25 at 4 inches with a +3.00 lens.

He was doing considerably better at school, no longer using the low vision aids, and having no difficulty seeing the blackboard or in activities with sports.

On July 26, 27, and 28, 1977 the child was taken off the diet and offered a number of foods known to contain synthetic colors, flavors and salicylates, *e.g.* dry cereal, powdered drinks, soft drinks, artificially colored ice cream and cookies.

No change was observed in eye movements until the child had been off the diet for two days. On August 1, 1977 examination of the eyes revealed vision had regressed to 20/200 in either eye separately and 20/100 together. Near vision dropped to 20/80. After being placed on dietary control it required approximately 21 days for complete restoration of vision.

The boy's nystagmus is under complete control so long as no infractions of the diet are experienced.

Case 2[3]. On March 29, 1978, a 5 1/2 year old female child was referred from ophthalmology because of nystagmus.

Pregnancy was normal as was the delivery. Birth date was November 29, 1971, and birth weight was 7 lb. 4 oz. An iron supplement tablet was the only medication during pregnancy. The mother does not smoke and took an alcoholic beverage about once monthly.

The child was breast fed for one year. At one month of age the mother first noted incoordination of the child's eyes; this was also observed by the pediatrician at repeated monthly examinations. Because of the persistent crossed eyes the child was seen by an ophthalmologist at four months of age. Crossed eyes were noted; the media was reported clear and fundus reflex pink.

At 10 months of age ophthalmological examination reported marked alternating esotropia with preferred fixation for the right eye. Pupils were normal; media was clear and normal. A diagnosis of alternating esotropia was made. Atropine drops 0.5 % t.i.d. for 2 days was ordered for purposes of refraction. The child was placed on glasses at 11 months, which had little effect upon the esotropia. Examination noted that the child holds fixation with the left eye for several seconds. At 2 1/2 years of age a patch was placed over the right eye for one month, then off for one month and on again for one month. This was continued until the child was 3 years of age.

At 4 years of age, no change was observed in the alternating esotropia, either with or without glasses; surgery was recommended and performed several months later.

[3] Referred by W. B. Shekter, M.D., Chief, Department of Ophthalmology, and Alice Byrnes, Certified Orthoptist, Kaiser-Permanente Medical Center, San Francisco, California.

On the 6th post-operative day the patient experienced diplopia which persisted for 15 days.

At the time of the initial examination in our Department 3/29/78 (5 1/2 years of age) the child was wearing glasses and for 2 weeks previously had worn a patch over the right eye. Nystagmus was very marked.

A report from the child's teacher in first grade stated the child was doing very well in all her academic work, actually above grade level, except that she was restless and talkative at times.

Enuresis had been present from birth.

A review of the child's diet diary revealed a high frequency of foods containing synthetic colors, flavors, BHT and BHA, as well as some thought to contain salicylates.

On April 10, 1978 dietary intervention was instituted, eliminating all artificial food colors, flavors, BHT, BHA and the list of foods thought to have natural salicylates (see Table 1).

One week after starting the diet enuresis cleared. The child showed some improvement in the eye condition, but a pronounced response

Table 1. *The Feingold diet*

Eliminate:		
Part I.	Synthetic colors and flavors contained in food Butylated hydroxytoluene (BHT) Butylated hydroxyanisole (BHA)	
Part II.	Selected foods with natural salicylates	
	Almonds	Peaches
	Apples (cider and cider vinegar)	Plums and Prunes
	Apricots	Tangerines
	Bananas	Cucumbers and Pickles
	Berries	Green Peppers
	Cherries	White Potatoes
	Currants	Tomatoes
	Grapes and Raisins (wine and wine vinegar)	Cloves
		Coffee
	Nectarines	All Teas
	Oranges	Oil of Wintergreen
Part III.	Benzoates*	
	Cranberries, Berries, Prunes, Greengage Plums, Cinnamon, Ripe Olives	
	Benzoates used as preservatives	

* All sources of benzoates must be eliminated in some cases.

was observed after omitting white potatoes and bananas, which have salicylates [7]. June 19, 1978, two months after initially starting the diet, a marked improvement in the eyes was observed.

September 3, 1978, approximately 5 months on the diet, the orthoptist reported a marked improvement in the child's perception as well as complete control of nystagmus.

September 7th to 12th, the child was challenged with a number of forbidden foods items, *e.g.* powdered drinks with artificial colors and flavors, apples, strawberry jam. A marked change in the child's behavior was noted; she became quite active, talkative and in conflict with siblings. Perception was impaired, but there was no evidence of nystagmus.

Discussion

As the hyperkinetic syndrome is defined today the two cases presented can be included in this category, even though hyperactivity was not a dominant feature of the clinical pattern. In Case 1 the child was subject to tantrums rather than hyperactivity; the second child was rather restless and talkative but not particularly hyperactive.

The syndrome of hyperactivity, at times labelled Minimal Brain Dysfunction (MBD), encompasses approximately 500 target deficits. The deficits occur in three categories: behavior, muscular incoordination, disturbances of perception and cognition. Not every deficit is observed in any individual child, rather each child has its own mosaic constituted of deficits from one or more categories [8]. Some children may have only a single deficit, such as impaired visual perception, auditory perception or hyperactivity. On the other hand, the child may not be hyperactive yet be included within the syndrome.

The clinical pattern is not only variable but also labile, so that the deficits observed in any child may vary from day to day and even from hour to hour. This great variability and lability is expressed in the literature by the great confusion regarding etiology, labeling of clinical patterns and management.

As has been indicated, a single compound apparently can produce in addition to hyperkinesis or MBD clinical patterns resembling those seen in a variety of neurologic disorders. It is perhaps the failure to integrate all of the signs with hyperkinesis or MBD under a single heading that eye muscle involvement manifested either as nystagmus or strabismus has not been emphasized as part of the hyperkinetic syndrome. However, on closer evaluation it becomes apparent that the oculomotor disturbances occur more frequently in conjunction with hyperkinesis than has been credited in the past. Prof. Jun

Tsutsui of Kawasaki University in Japan reports (personal communication) that of approximately 500 children with strabismus no less than 30 percent are also hyperactive. It would perhaps be advisable to encompass the clinical patterns of this disorder in a single rubric, such as neurologic or neuromuscular disturbances.

We currently have five additional cases under observation who are being studied more precisely for their response to controlled, blind dietary management to evaluate the possibility of a placebo effect. These children also have nystagmus, disturbed perception and variable degrees of behavior and learning disabilities.

The response of the cases reported and those under observation suggest that food constituents may be extremely important in affecting the eye muscles as well as visual perception.

In the dietary management of eye disturbances we have been guided by the experience gained from the management of behavioral disturbances and learning disabilities. The diet listed in Table 1 may be effective for control of behavior in about 60 to 70 percent of cases. For the remaining 30 to 40 percent we must search elsewhere, since any compound in existence, either natural or synthetic, may induce an adverse reaction in any individual with the appropriate genetic profile, or predisposition. In the management of behavior we occasionally encounter individuals who were intolerant to milk or at times corn, wheat, etc. It is conceivable that a similar situation may be operating in eye muscle disturbances. However, in the management of the seven eye cases we have observed to date, we are impressed with the importance of foods containing a salicylate radical (see Table 1). In addition to the items listed we have observed that white potatoes and bananas, both of which are a source of salicylates, must be eliminated. In the first case reported, benzoates, which occur in most berries [9], many fruits [10] but particularly cranberries, seemed to induce nystagmus.

To date we have managed nystagmus only in children with behavioral disturbances. It is possible that in some cases of eye involvement overt behavioral disturbance may not be observed. For such individuals a diagnostic trial with the elimination diet is recommended. The diet that we use is fully nutritious and carries little risk.

In every instance we have observed that with improved behavior the prognosis is usually good. This observation also holds true with children who have learning disabilities complicating the pattern. In our experience if behavior responds, other deficits will clear. It may require a longer period of observation—2 weeks to several years; however, with patience the deficits will improve.

Here again, our experience with eye muscle involvement is not sufficient to permit absolute conclusions. We have observed that upon challenge of children with nystagmus the condition does not respond immediately, as frequently observed with behavior. In Case 1 three days of a heavy load of items listed in Table 1 were required before nystagmus returned, with a loss of visual acuity. In Case 1, as indicated, 21 days were required for a washout to establish a normal baseline. Judging from our experience with behaviorally disturbed children, these figures may vary from child to child.

References

[1] Report at the annual AMA meeting in New York, June 1973 and in June 1974 at the annual AMA meeting in Chicago.

[2] Thousands of letters received from all parts of the United States, as well as from Canada, Australia, New Zealand and Norway have reported favorable responses to the diet for management of behavioral disturbances and learning disabilities.

[3] National Advisory Committee on Hyperkinesis and Food Additives. Report to the Nutrition Foundation, June 1, 1975.

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(b) Food Additives Project. Kaiser-Permanente Medical Care Program and the University of California at Berkeley. (Personal communication.)

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[10] *White, Handler, Smith, Stetten:* Principles of Biochemistry, p. 776. New York: McGraw-Hill. 1954.

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