Treatment of a Mucopolysaccharide Type of Storage Disease with the "U" Series

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Abstract

There are at least four subtypes of mucopolysaccharidosis III or Sanfilippo syndrome (Neufeld, 1980), a group of autosomal recessive lysosomal storage diseases caused by defective heparan sulfate degradation (Harris, 1961; Sanfilippo et al., 1962; Sanfilippo et al., 1963). The present case represents a possible fifth subtype of Sanfilippo syndrome or a different MPS disease. The prototype of the mucopolysaccharidoses is Hurler syndrome (MPS 1-H), described by Hurler (1919) two years after Hunter recognized the Hunter syndrome, MPS II. There appear to be severe and mild forms of all these diseases.

Each of the subtypes of Sanfilippo syndrome is associated with a different enzymatic defect: Sanfilippo A (heparan N-sulfatase), MPS III-B (alpha-N-acetylglucosaminidase), MPS III-C (acetylCoA: alpha glucosaminide N-acetyltransferase), MPS III-D (N-acetylglucosamine 6-sulfate sulfatase) (Klein, Kresse, von Figura, 1978; von Figura, 1980). Whether because of insufficiently sensitive chemical tests, or because these substances are not the actual intermediates in normal MPS degradation, abnormal metabolic pathways having been activated (Neufeld, 1973), mucopolysaccharidiuria of Sanfilippo A cannot be distinguished from mucopolysaccharidiuria of Sanfilippo B, and so on (Horwitz, 1979). Heparan sulfate accumulates in all structures. Physical abnormalities include coarse facial features, macrocephaly, and hepatomegaly. Even within the same family, in which three of six siblings were affected with Sanfilippo B, there were significant variations in clinical features, including the absence of relevant skeletal and joint abnormalities in one of the severely affected siblings, and fluctuating mucopolysaccharidiuria, ranging from very high levels to trace amounts, in the sibling with mild mental retardation (Andria et al., 1979). Despite such variations in the clinical picture, severe neurologic anomalies are common. There is steady neurological deterioration, and life expectancy does not exceed twenty to thirty years.

The proband, born April 24, 1975, was diagnosed as a patient with Sanfilippo syndrome on the basis of urine studies positive for mucopolysaccharides, predominantly

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FIGURE 1

ADULT DOSAGE OF THE "U" SERIES PER CAPSULE OR TABLET

UMORPHOID – A & B	BREAKFAST
Thyroglobulin	66 mg.
L. Triiodothyronine	25 mcg.
Organic Iodide	66 mg.
Vit. A (water dispersible)	25,000 I.U.
Vit. E as d-Alpha-tocopherol Acid Succinate, N.F.	10 I.U.

UPNEOID	B.L.D.
Phenylpropanolamine Hydrochloride	20 mg.
Pyrilamine Maleate	25 mg.
Rutin	20 mg.
Ascorbic Acid †	100 mg.
Theophylline Magnesium (or Sodium) Glycinate	100 mg.

† ascorbic acid to be individually supplemented.

UTEPTOID A	BREAKFAST–DINNER
Betaine-Choline Tartrate	100 mg.
Choline-Methionine Tartrate	100 mg.
Inositol	50 mg.
Unsaturated Fatty Acids (linoleic-linolenic-sitosterol)	100 mg.
Desiccated Liver	150 mg.

UPEPTOID B	DINNER
Betaine HCl.	66 mg.
Papain	66 mg.
Pepsin	66 mg.
Pancreatin	66 mg.
Diastase	3.3 mg.
Ketocholanic Acid	66 mg.
Desoxycholic Acid	66 mg.

UNOID	B.L.D.
Pentylenetetrazole	50 mg.
L. Glutamic acid	200 mg.
Nicotinic acid	50 mg.

Full dosage . . . . . ten years and over
Half dosage. . . . . ages 5-10 years
One-third dosage. . . . . ages 2-5 years
One-fifth dosage . . . . . ages 1-2 years
One-tenth dosage . . . . . under one year old

UTROPHOID B	LUNCH
Thiamin Mononitrate	20 mg.
Riboflavin	20 mg.
Calcium Pantothenate	20 mg.
Para Aminobenzoic Acid	20 mg.
Pyridoxine	20 mg.
Niacin	20 mg.

UTROPHOID C	DINNER
Cyanocobalamin	25 mcg.
Folic Acid	5 mg.
Calcium (Calcium citrate)	30 mg.
Cobalt (Cobalt Chloride)	0.1 mg.
Copper (Copper Sulfate)	1 mg.
Iodine (from Potassium Iodide)	0.15 mg.
Iron (ferrous gluconate)	10 mg.
Magnesium (from Magnesium Sulfate)	1 mg.
Manganese (from Manganese Sulfate)	1.25 mg.
Molybdenum (from Sodium Molybdate)	0.1 mg.
Zinc (from Zinc Sulfate)	1 mg.

SUPPLEMENTS	
Zinc gluconate	31.5 mg.
Calcium Pantothenate	50 mg.
Pyridoxine	50 mg.
Potassium Gluconate	50 mg.
Magnesium	50 mg.
Niacinamide	15 mg.
Thiamin	2.2 mg.
Riboflavin	2.2 mg.

UPNEOID C	PRN
Naphazoline HCl.	0.025%
Pyrilamine Maleate	0.250 %
Chlorpheniramine maleate	0.125%
Methyl Paraben	0.005%
Propyl Paraben	0.010%

DENTAL ANESTHETIC	PRN
Benzocaine USP	16%
Chlorobutanol USP	5%

DIURETIC

Furosemide 40 mg. - two days per week (e.g. Sundays and Thursdays) with breakfast only.

	BREAKFAST–LUNCH
Bone meal contains: Phosphorus	300 mg.
contains: Calcium	600 mg.
contains: Vitamin D-3 (natural)*	100-200 I.U.
Calcium 130 mg./Magnesium 60 mg. from Dolomite	200 mg.

* represents the natural form of vitamin D3 contained in bone meal, or if less than 400 IU would be available daily, natural fish liver oil D3. No crystalline D3 and no form of D2 are ever added to the "U" Series.

heparan sulfate, showing a value of 600 with an upper normal limit of 220. He was referred to the Genetics Department of the University of Michigan, which confirmed mucopolysaccharidiuria, delayed turnover of mucopolysaccharides in tissue culture cells, accumulation of radioactive sulfate in tissue culture, and the clinical picture of macrocephaly with mild skeletal changes and liver palpable about 3 cm below the right costal margin. His I.Q. on the Stanford Binet, form L-M, was 81. The University of Michigan referred him for treatment with the "U" Series (Fig. 1), an orthomolecular approach to the treatment of storage diseases (Turkel, 1975). He was first treated May 21, 1979, at the age of 4.1. Clinical improvements that coincided with treatment were rapid and dramatic, according to his physician at the University of Michigan (Wilson, 1980). Biochemical changes also occurred. Within four months of treatment, the urinary MPS (HS) count fell to 200. After ten months of treatment, it was 90, leading the National Institutes of Health to suggest a revision of the diagnosis.

CASE HISTORY

Birth History: Clark was born April 24, 1975. He was the first child of the 22 year old father and the third child of the previously married 33 year old mother. Pregnancy, complicated by toxemia, was full term. Delivery was extremely difficult because of the infant's very large head, circumference 38 cm. A spinal anesthetic was given, and forceps were used to deliver the 9 lb., 12 oz. infant. A "battered" appearance and neonatal jaundice of unknown origin were noted.

Family History: Macrocephaly, mild mental retardation, and lipomatosis are hereditary conditions seen in the paternal side; father, grandmother, and two surviving uncles are affected. A third paternal uncle died at the age of eight months of an unspecified condition associated with macrocephaly. A maternal cousin has an enlarged head. A maternal half-brother had died before Clark's birth, of SIDS at the age of eight months. A urine screen for mucopolysaccharides of other family members proved negative.

Developmental History: Milestones were delayed. Clark lifted his head at eight months, rolled over at eighteen months, and sat up at two years. At the age of three, his locomotion was clumsy and ataxic. During a neurological examination, he stumbled, seemed to lose his balance, and bumped into furniture on three separate occasions. He held his arms, from his elbows to his fingers, flexed in a high adducted position. He slid down steps and crawled up. A visual-perceptual problem, dysphrasia, and dysphagia were noted. His inability to chew properly, with subsequent choking on food, was life threatening, requiring emergency treatment on several occasions. He sweated profusely at night and ground his teeth. His eyes seemed to be only half closed while he slept.

At age 3.0, he was tested with the Stanford Binet, L-M, and Denver Developmental Screening Test. On the Stanford Binet he attained an I.Q. of 81, which placed him in the low average range of intelligence. On the Denver Developmental Screening Test he attained a developmental age level of 2.3 in the personal social area, 3.0 in fine motor adaptive, 2.3 in language, 2.0 to 2.3 in gross motor.

Speech and language evaluation was made at age 3.2. Verbal ability was tested with the Peabody Picture Vocabulary Test-A. Mental age equivalent was 2.1. On the Utah Test of Language Development, his language age equivalent was 2.2. According to the examiner, Clark manifested a significant delay in both understanding and use of language as a meaningful communicative tool.

Physical findings: Clark's head circumference at three weeks was 41 cm. At seven months he was 30 inches long, weighed 20 lbs., 8 oz.; his head circumference was 51 cm; the anterior fontanelles were some four to five inches in length, open and bulging. Hydrocephalus was ruled out; studies demonstrated no obstruction, and there was no increase in cranial pressure. Tests for dwarfism and thyroid deficiency

proved negative. A podiatrist prescribed corrective shoes for platypodia associated with hyperflexibility of joints. Ongoing problems included allergies, as well as asthma, treated with Sudafed and Quibron; dysphagia for all but semi-liquid foods, leading to choking and cyanosis; and macrocephaly, etiology unknown. Hydrocephalus, dwarfism, and hypothyroidism having been ruled out, further studies were planned to rule out megalencephaly of DeMyer, mucopolysaccharidosis, and other genetic syndromes.

Diagnosis: Clark's urine was sent to Upjohn Laboratories by Beaumont (Michigan) Hospital. His urine was positive for mucopolysaccharides (June 28, 1978). However, a skeletal survey failed to reveal radiographic evidence of a mucopolysaccharidosis in the spine, long bones, ribs, or hands. The sella turcica was intact with a very slight omega-shape. A second urinalysis, July 25, 1978, again was positive for mucopolysacchariduria. A skin biopsy was positive.

Clark was referred to the University of Michigan for further evaluation. A third urine screen taken January 29, 1979 was positive. A skin biopsy confirmed that the cells as well as urine accumulated heparan sulfate. Clark's fibroblasts were compared with those of a normal control and a patient with alpha-L-iduronidase deficiency (MPS I). In the control, approximately 50 percent of MPS was degraded, as compared with 20 percent in the MPS I patient, and no degradation of MPS in the Clark fibroblasts. There was a delayed turnover of radioactive sulfate in tissue culture: compared with three normal controls (7 to 12.9 hour half lives), Clark's turnover of radioactive sulfate had a half life of 20.4 hours. No accumulations of chondroitin sulfate or dermatan sulfate were found in these examinations of the fibroblasts. Since exclusive storage of heparan sulfate is a characteristic feature of MPS III (Kresse, von Figura, and Klein, 1978), the diagnosis, based on clinical signs, a typical phenotype, and five positive laboratory studies demonstrating storage of heparan sulphate, was that Clark's disease was one of the types of MPS III, Sanfilippo syndrome.

Therapeutic intervention: April 25, 1979, when he was four years old, he was referred by the University of Michigan for treatment with the "U" Series. He was examined physically and radiographically on May 21, 1979. His bone age was three to four years. Height was 43¾ inches, at the 95th percentile; his weight at 43¾ pounds, was in the 90th to 95th percentile. His heart was within normal limits; lungs were slightly congested. There was splenomegaly, macrocranium, flattening of the acetabular angle with flaring of the iliac wings. The liver was palpable 3½ cm below costal ribs. Pulse was 100, respiration 18. His head circumference was 59 cm with some brachycephaly (10 cm) and frontal bossing. There was broadening and flattening of the nasal bridge. His skin was slightly puffy and marmorated. His hair was fine and fair. Hirsutism was present, especially on the back. There was considerable cerumen in both ears. His vision was normal, and there were no opacities. His gum line was thick; the palate was narrow and appeared to be high. His tongue was normal. His abdomen protruded. The first and second toes were widely spaced and his arches were flat. His muscles were hypotonic. The joints of his hips, knees, feet, elbows, and especially of shoulders and fingers, were hypermotile. His mental age on the Peabody Picture Vocabulary Test was 2.11, and his social age on the Vineland Scale was 2.2. He was enuretic and could not move his bowels without the help of enemas.

August 1979, he was reevaluated by his neurologist, who again confirmed the diagnosis of Sanfilippo syndrome. September 18, 1979, he was reexamined at the University of Michigan. According to the report, he still had the appearance of macrocephaly. His chest, heart and abdomen were normal with no sign of organomegaly. He seemed more alert and vigorous, and his nutritional status appeared more robust. The impression remained Sanfilippo syndrome, based on delayed turnover of mucopolysaccharides, mucopolysaccharidiuria, and skeletal changes. Since specific measurement of enzymes deficient in types A and B demonstrated no abnormality, it was suggested that Clark represented either an untested

type, or a new type that had not been previously recognized.

When reexamined at our office December 18, 1979, he was healthy and sociable. He was said to be participating in neighborhood activities and playing with other children. His mental age was 3.9, a gain of ten months in mental age during the seven month interval between examinations. He spoke more clearly and had a larger and more complex vocabulary. Physically, he was more flexible and mobile. He ran less awkwardly. His lungs were clear, respiration 18, heartbeat regular, no murmurs, rate 96. His digestion was much improved, though he remained slightly constipated. His liver was no longer palpable. His skin was marmorated and rough. His height was 44 inches, weight 44 pounds, subcutaneous tissue 15 mm. His head circumference was 59 cm, brachycephaly 9 cm; fontanelles were closed.

March 5, 1980, he was examined at the University of Michigan Genetics Clinic. Cells were taken from Clark's back for study. According to his physician, Clark seemed more robust, alert, and vigorous, coinciding with the "U" Series therapy. Blood was drawn at the University of Michigan March 5, 1980 for additional thyroid studies, and in order to prepare serum to be sent to the NIH for further evaluation on March 24, 1980. The University reported that the T_4 was within the normal range, and the T_3 was slightly below normal levels. Continued evaluation of the familial lipomatosis and thyroid function was planned.

No deterioration occurred during the time of treatment with the "U" Series. Instead, his total condition was greatly improved; however, when medication was discontinued on a trial basis for a week, he again became lethargic. By June 18, 1980, he was 46¼ inches tall, and he weighed 49 pounds; he was considered mentally normal and was healthy. He had no serious interval illnesses, no allergies. To date, this child, who had been said to be dying, continues to thrive.

Within nine months of treatment, his I.Q., evaluated by the Intermediate School District, rose from 81 to 113. His physician at the University stated that he was amazed at the physical and behavioral improvements. Urinary MPS (HS) count dropped from 600 to 424 to 200, and at the March 5, 1980, examination, to 90. Pretreatment studies of Clark's urine and cells revealed the presence of an MPS storage disease. After a few months of treatment, the accumulations of heparan sulfate disappeared from the urine and the patient's condition improved dramatically.

The unanticipated improvement that coincided with "U" Series therapy puzzled Clark's doctor at the Genetics Department of the University of Michigan. March 24, 1980, he sent frozen serum, together with a summary of the events of Clark's life, to the National Institutes of Health. The summary included the genetic information regarding macrocephaly and multiple lipomas, slow mental development, the death of an uncle "early in life of questionable 'water on the brain'," the consistently high values of MPS prior to treatment, and a positive test for radioactive sulfate incorporation by his cells, "showing that his turnover of mucopolysaccharides was abnormal."

The letter to the NIH further stated that Clark "seemed dramatically improved on this ["U" Series] regimen. His general level of alertness, vocabulary, strength, and activity seem substantially better than a year ago. He is not completely toilet trained yet, but he is making progress in that area also. We repeated a mucopolysaccharide screen on his urine, and it was normal, showing a value of 90 compared to an upper limit for normal of 220." Surgery to excise a fatty tumor in the rectum was performed April 14, 1980, with no complications.

May 6, 1980, Clark was observed by physicians at the NIH. The conclusion was that the "diagnosis of Sanfilippo was most certainly incorrect, an unfortunate result of a fluctuation in a laboratory value, and has been revised." Accordingly, the University of Michigan prepared the following revision July 9, 1980: "We feel that Clark's excellent development over the past two years is inconsistent with the diagnosis of mucopolysaccharidosis...He seems to have a genetic syndrome which involves a large head and a propensity to develop certain types of benign fatty tumors. His improvement has co-

incided with the controversial vitamin therapy he has received from Dr. Henry Turkel, although we are not certain which of these medications may have benefited Clark."

That Clark was no longer considered to be a retarded child, and that his condition was no longer considered to be lethal, was naturally a source of gratification. However, the new diagnosis of a benign, previously unknown hereditary disease that closely mimicked characteristics typical of a fatal, untreatable, neurologically devastating disease, left his mother with many questions about his present condition and its prognosis, including the significance of the diagnostic levels of heparan sulfate in his urine and cells prior to treatment. The NIH studies of his fibroblasts after treatment revealed MPS accumulations in the high-normal range. She queried the NIH and the University of Michigan regarding other diseases associated with macrocephalus, neurological disturbances, mental retardation, and fluctuating levels of heparan sulfate in the urine and fibroblasts.

The NIH suggested that Clark was a child with cerebral palsy, and recommended speech therapy. The University of Michigan responded that other possible causes for this constellation of disorders could be dwarfism and stress. These three alternative diagnoses fail to conform to the facts of this case. The final suggested diagnosis, a genetic syndrome involving a large head and benign fatty tumors, a rare disease recognized in five families in the United States, fails to account for the presence of heparan sulphate in Clark's urine and cells before treatment with the "U" Series.

The frozen serum and cells, prepared for the NIH by the University of Michigan March 5, 1980, ten months after the start of treatment, were tested for mucopolysaccharide storage disorders, and the enzymes that would have been missing if he had any of the four unknown forms of Sanfilippo diseases were measured. All four known forms of the disease were ruled out by these studies, in August, 1980.

Note:

Patient Clark's I.Q. on the WISC-R (administered by the school district 7/8/81 and 7/22/81) was as follows:

Verbal I.Q.	107
Performance I.Q.	136
Full Scale I.Q.	123

Before treatment with the "U" Series, "his I.Q. as measured by the Stanford Binet, Form L-M was 81 [at age 3.0]." After treatment, at age 4.11, his full scale I.Q. on the WPPSI was 113. Presently, on the WISC-R, the full scale I.Q. is 123.

DISCUSSION

Therefore, Clark represents either a new mucopolysaccharidosis syndrome, a fifth subtype of Sanfilippo syndrome, or a previously unknown hereditary disorder associated with fluctuating levels of MPS, hereditary macrocephaly, mental retardation, disabilities in swallowing and articulation, mild ataxia and lipomatosis. Since it is the function of the "U" Series to remove accumulations, whether this patient's condition, associated with accumulations of MPS, is unique to his family or is an MPS storage disease, the amelioration of his condition—namely the dramatic improvement in his health, intellectual abilities, and biochemistry—is due to treatment with the "U" Series. We propose several alternative suggestions regarding the therapeutic effect of the "U" Series. As in all storage diseases, including Down's syndrome, the accumulations of the unconverted substrate interfere with normal utilization of nutrients; therefore a portion of the retardation problem is due to malnutrition. Malnutrition delays development of the entire body. After the child's birth, the underdeveloped excretory organs function inefficiently. Wastes (which are water-soluble substances, fat-soluble substances, and minerals) are incompletely eliminated, and their retention causes further accumulations and retardations. Untreated, the condition worsens (Figure 2 represents all storage diseases. Figure 3 represents all trisomic syndromes).

Two patients with a firm diagnosis of Sanfilippo syndrome (MPS III-A) have started "U" Series therapy within the past three months. Billy, born March 29, 1969, had deteriorated and could no longer speak, was no longer toilet trained, and screamed almost con-

FIGURE 2

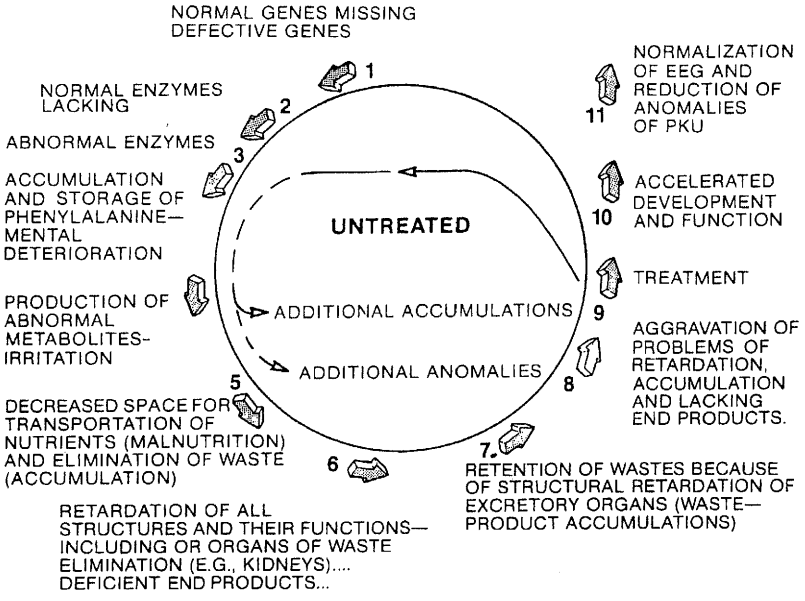
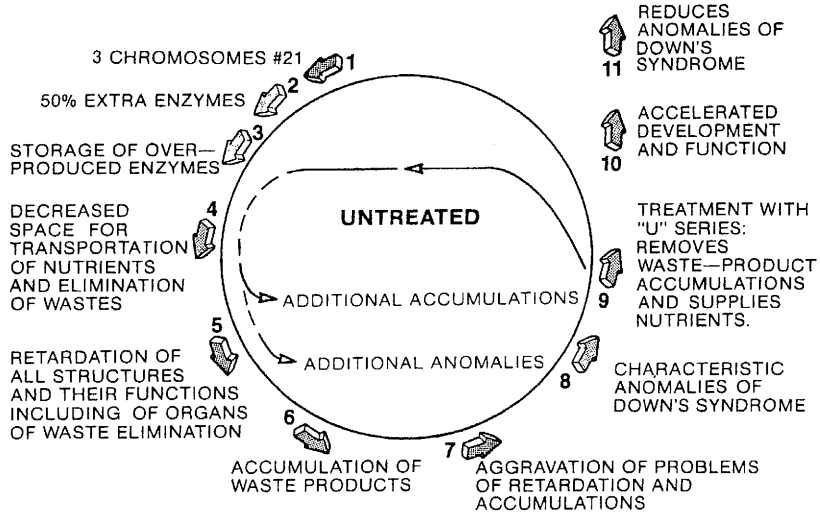


FIGURE 3



BREAKING THE CYCLE OF DEVELOPMENTAL ANOMALIES OF DOWN'S SYNDROME WITH THE "U" SERIES

stantly. His attention span is increasing. He can now watch television with his family for five to ten minutes at a time. Chris, born June 21, 1975, was more mildly involved. His bone survey was normal except for a bone age of 4.6 at a chronological age of 5.7. He has started to speak about events that happened in the past, which he had not previously been able to do. At this early stage of treatment, both families remain cautiously optimistic.

The "U" Series, designed to help eliminate various accumulations that result from incomplete metabolization of substrates and inefficient removal of waste products in genetic diseases, and simultaneously and synergistically to accelerate growth and development of the retarded structures and their functions, can ameliorate all types of inborn errors of metabolism (Turkel, 1980), including those associated with borderline retardation (Turkel and Nusbaum, 1978) and cataracts (Horwitz, 1981).

In the case of storage diseases, such as the mucopolysaccharidoses, the "U" Series aids in removal of some MPS accumulations and in simultaneous supplementation of nutrients that function as co-enzymes, accelerating general development. The genetotrophic theory proposed by Williams (1956), and successfully tested by Harrel, Capp, Davis, Peerless, and Ravitz (1981), also helps to vindicate the rationale of nutritional components within the "U" Series. Since vitamins act as co-enzymes in numerous metabolic steps, enhancing the patient's nutritional status may improve the function of deficient metabolic pathways or set up alternative pathways of MPS degradation.

Specific therapy is not being vigorously pursued for these diseases because of the problems of "1) getting the enzyme to the target organ, such as the brain, and 2) preventing rejection of the foreign enzyme by the body. Since these problems are common to almost all storage diseases, solving them for one such disease will facilitate treatment of others" (Vidgoff, 1979). The enzyme is not being rejected, but it is also not accepted. The accumulations that interfere with its entry to the target site prevent its acceptance by the body. Removal of some of these accumulations may eventually solve the problems of enzyme replacement therapy, not only for the mucopolysaccharide storage diseases, but for other storage diseases such as the sphingolipidoses, in which the damaging accumulations of sphingolipids, especially of Tay-Sachs disease, may prevent the appropriate enzymes from reaching the target site (Henahan, 1971).

AN ADDITIONAL STUDY

Cataract Dissolution in a Patient with Down's Syndrome

Underlying Rationale: Down's Syndrome, like other metabolic disorders, may cause lenticular or corneal opacities (Eissler and Longenecker, 1962; Cullen and Butler, 1963). Fine lens opacities visible with slit lamp biomicroscopy have been reported in 59 percent of patients with Down's syndrome, and cataracts have been reported in 1.3 percent (Smith, 1976). Diseases associated with metabolic accumulations may lead to retention of water-soluble subtances in the lens and disruption of its normal structure and metabolism. By gradually reducing accumulations, "U" Series therapy has significantly improved the vision of Down's syndrome patients.

L.B., born May 23, 1959, spoke at age three, and was toilet trained at four and a half. By age seven, he was regressing. At eight he was placed on Mellaril. When he was ten, the Ohio Medical College ophthalmologist told his mother that for all practical purposes, L.B. was blind because of bilateral cataracts. He entered Columbus State Institution at the age of 15.

L.B., a typical Trisomy 21 patient, was first examined April 28, 1975. He had bilateral cataracts and gave no indication that he was able to see. A variation of the "U" Series was dispensed. Pentylenetetrazole, nicotinic acid, and glutamic acid were eliminated until May 17, 1977. Bromolain, 80 mg TID and papain enzyme 21.34 mg TID were added. This medication was first administered to L.B. at the Institution on July 28, 1975, but was given only sporadically by the reluctant staff until after L.B.'s transfer to a residential center in November, 1979.

Ophthalmological Summary from the Medical College of Ohio

6/11/71 Intumescent cataract OD.
7/2/71 Does have a cataract OS.
2/25/72 Cataract OD Intumescent; cataract OS moderately advanced.
9/6/73 Dense cataract OD - maturing cataract OS.
6/6/74 Picks up small objects from hand. Bilateral cataracts OD OS.

Intumescent cataract OD, probable posterior subcapsular cataract OS.

Plan no surgery until cataract OS becomes mature.

(Treatment started 6/28/75.)

1/30/76 Dense mature cataract OD, posterior subcapsular cataract OS.

Cataract extraction recommended on right eye since left eye will follow suit in time.

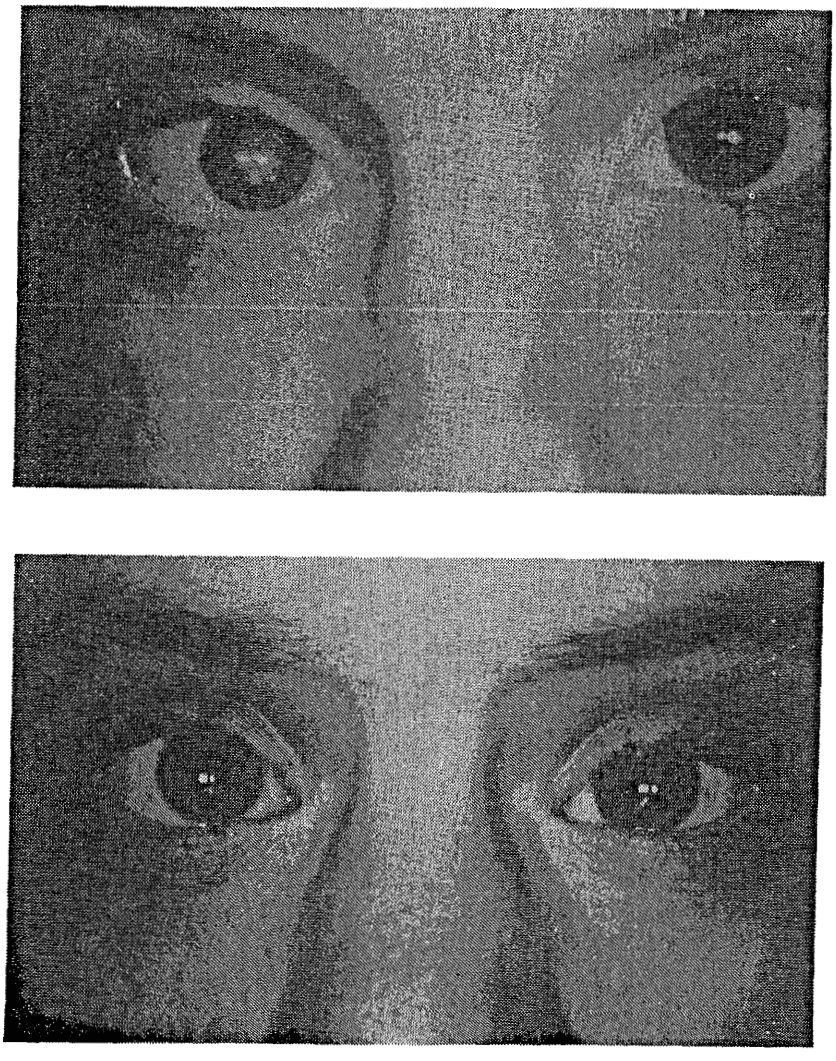
5/7/76 Intumescent cataract OD; OS dot-like opacities. Surgery not wanted or needed.
8/2/77 Dense cataract OD; nuclear cataract OS. Mother thinks he is seeing better recently.
6/19/80 OD - 20/20; OS - 20/100. Impression: Rupture of mature cataract OD ? Traumatic ? Cataract OS.

There was no trauma or sign of rupture.

9/28/81 Spontaneous resorption of cataract OD—No damage to lens, early posterior subcapsular cataract OS.

Photographs demonstrate dissolution of the cataracts. (Figure 4).

FIGURE 4



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