Megavitamin Therapy for Different Cases

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ASCORBIC ACID CURE OF ONE CASE OF TOXIC PSYCHOSIS

Recently when I was sorting out my reprints on ascorbic acid I ran across a complete file on my first psychotic patient given large doses of ascorbic acid alone. I have referred to this case elsewhere, but have not given any clinical detail. Since this may well be the first case ever treated in this way, I think it may be useful to report the details.

Mrs. V. H., age 47, was referred to the General Hospital, Munroe Wing, Regina, because she was delusional, very depressed, and threatened to kill herself. She was admitted December 9, 1952.

She was large, obese, masculine in appearance with a lot of hair on her face. Her complexion was pale and muddy. She suffered from perceptual changes, hearing the voice of her priest, she was confused, delusional, with no insight, and was very agitated, restless, and depressed with marked mood swings.

About four years before her jealousy of her husband suddenly became intense and she accused him of having an affair with his secretary. She then threatened to kill herself. One year before she was admitted she had her right breast removed as treatment for cancer and then received follow-up radiation. The skin over her breast ulcerated. About five weeks before admission she was given testosterone until 10 days before.

Because of her severe agitation, depression, and paranoid delusions, ECT, the main treatment of that day, was indicated.

By December 11, 1952, I had persuaded her doctor to allow me to treat her with megadoses of ascorbic acid, my reasoning being that she was probably toxic from her cancer, radiation, and the ulceration. He agreed to hold off ECT for a few days.

She was started on 1 g of ascorbic acid each hour while awake. If sleeping she was not awakened, but when she did awaken was give 1 g for each hour she had slept.

Progress Under Treatment

Between her admission December 9 and the first administration of ascorbic acid there was no change in her psychosis. Ascorbic acid was started 2:30 p.m., December 11, 1952 (Thursday). The following information is taken from the nursing notes:

[1] **#3 2727 Quadra St., Victoria, B.C. V8T 4E5.**

(1) December 12, 1952:

A.M. —Patient slept eight hours. She ate a large breakfast, stated she felt better, but complained of feeling weak and of pain and burning over her ulcerated breast area. She carried her Bible with her, praying frequently. The pain in her breast, she stated, was there because someone was praying for her to be punished.

P.M. —The patient was more relaxed. She was friendly and affectionate with the nurses, but required frequent reassurance that she would recover. She could not concentrate on anything.

(2) December 13, 1952:

A.M. —Patient was cheerful but quiet and no longer sought reassurance. The ulcer had begun to heal. Patient participated with the group and enjoyed singing Christmas Carols with the radio. The ascorbic acid caused frequent bowel movements.

P.M. —Patient remained cheerful and quiet.

(3) December 14, 1952:

Patient remained the same. She was seen encouraging another patient who was very upset. She missed her husband and was excited and happy to see him. She did not carry her Bible with her this day.

(4) December 15, 1952:

She remained mentally well. She told the nurses, "I like Dr. L. better now and I really don't mind being here as I'll be getting better." Later that day she felt well enough to go home. Ascorbic acid was discontinued this day.

(5) December 17, 1952:

The patient stated she felt really good. For the first time her breast ulcer was clean and healing with no more purulent discharge.

(6) December 20, 1952:

The patient was discharged December 20 mentally normal. She remained normal until she died six months later from her cancer.

Her psychiatrist recorded the following notes:

"December 15, 1952—Patient started on ascorbic acid December 11. This day she sang hymns and was fearful she would be harmed. On December 12 she was much more at ease, mixed with other patients, and played cards well. On December 15 she was cheerful, no longer expressed delusional ideas, and reported she was much better. On December 17 she remained well. She described how terrified she had been prior to admission. People seemed to have changed and some appeared to be turkeys or dodos with their intestines hanging out. She had felt everyone was going to be killed, but now she felt it was strange she could have felt the way she did." There was no longer any indication for ECT and she was discharged.

When I had first seen this patient I had considered she had a toxic psychosis, but the director of the unit to whom Dr. L. reported concluded she was schizophrenic. I still believe I was correct. I am also convinced that the massive doses of ascorbic acid promptly brought her out of her psychosis and also initiated healing of the ulcerated lesion. This had not responded to any previous treatment. If she had not responded she would have been given a series of ECT. Tranquilizers were then not available. Improvement was noted within 24 hours.

One could make the improbable hypothesis that she had made a placebo response. But patients who have a toxic psychosis are not good candidates for placebo response. No attempt was made to convince her the vitamin would help since none of the staff had any reason for believing it would work, nor was she my patient at any time, nor has it been found that faith can produce such a rapid response to a serious infected radiation-induced ulcer.

I am sorry I did not keep her on ascorbic acid much longer in view of the recent work by L. Pauling showing that megadoses of this vitamin had been helpful for some patients dying from cancer.

MEGAVITAMIN TREATMENT FOR PSYCHIATRIC CHANGES

INDUCED BY MERCURY POISONING

Minamata disease, the result of chronic mercury poisoning, has become a serious problem in a few areas of Canada populated chiefly by native people. Mercury dumped into the water system contaminated fish which when ingested causes an increase in mercury levels. The disease is named after a Japanese town where it was first clearly recognized and described.

Chronic exposure to mercury has long been known to produce changes in the central nervous system. In the past the main exposure has been to inorganic mercury used in industry, but more recently organic mercury has become more important. There are differences in the way the body deals with organic and inorganic mercury. The main difference is that organic mercury compounds penetrate more readily into the brain, achieve higher CNS levels, and are excreted more slowly.

Apparently there is no effective treatment for Minamata disease. Perhaps one might develop. Since 1969 I have been confronted with the necessity to treat three men who had suffered inorganic mercury poisoning from prolonged exposure to metallic mercury. One recovered, and the other two were much improved and on the way to full recovery when last seen. This suggests that a similar approach might be therapeutic for some sufferers from Minamata disease, especially if started early.

The Three Cases

Case No. 1, **Born in 1945, Male:**

When first seen early in 1969 the patient complained of excessive nervousness for the previous three months. Just before this happened he had come into much closer contact with mercury in his laboratory where he worked as a chemist. No provision had been made for proper ventilation of the laboratory, and he did not do the preparations in a fume cupboard. With his nervousness he found himself more and more depressed, and he began to experience abnormal sexual impulses which led to antisocial behavior. About 17 days before he was seen he had been transferred to another line of work and was no longer exposed to mercury. By the time he saw me he was somewhat better.

A few months before he was seen he had a transient rash on his legs lasting one week which was itchy, and he noted he was very thirsty and his gums began to bleed when he brushed them.

His mental state was somewhat paranoid. He felt people were watching him continually, was convinced that his supervisor was talking about him and was intentionally trying to harm him. There was blocking, he could not remember as well and could not concentrate. He could read, but could not comprehend what he read. On occasion he suffered from insomnia.

In Cecil's Textbook of Medicine, Ninth Edition, the symptoms of mercury intoxication included:

(1) Stomatitis with salivation, a metallic taste, and a red-brown discoloration and gingivitis.
(2) Increased irritability, increased shyness, insomnia, headache, vertigo, depression, and rarely hallucinations.
(3) Tremor of muscles of the face and limbs.
(4) Loss of appetite and diarrhea.
(5) Dermatitis, erythematous in form, and desquamation.

The clinical picture, plus his exposure to mercury vapor, suggested he was poisoned by mercury. A blood specimen was sent to the laboratory. His mercury level was very high. Since the laboratory had hitherto run very few mercury assays, it was not certain of the exact amount.

My final diagnosis was that he suffered from an acute schizophrenic syndrome caused by mercury intoxication. As it was

not very severe, I started him on nicotinic acid, 1 g, q.i.d., since this is one of my main treatment components for schizophrenia, and on ascorbic acid, 1 g, t.i.d. Ascorbic acid has been shown to decrease the toxic effects of some heavy metal poisoning including that from mercury. He was also given Diazepam, 10 mg, q.i.d., for about one and one-half months.

He improved very rapidly and was almost normal one and one-half months after treatment was started. The Diazepam was discontinued, but he remained on vitamins. When finally seen three months after his initial interview, he was normal, but properly concerned over his previous behavior which he now was aware had been very abnormal.

Follow-up evaluation included the HOD test (Kelm et al., 1967; Hoffer and Osmond, 1961). This is a card-sorting test designed to measure the experiential world (sensory) of patients. Schizophrenics have high scores while normal subjects have low scores. His scores are shown on the table below.

	Total	Perceptual	Paranoid	Depression	Short Form for Schizophrenia
March 14	83	13	5	14	5
March 24	74	15	4	7	8
April 28	3	0	0	0	0
June 4	3	0	0	1	0
Normal Range	0-30	0-3	0-3	0-3	0-1
Mean for Schizophrenia	65	8-10	6-8	7-9	over 2

Since then he has not sought further help, was no longer exposed to mercury vapor, and presumably remained well.

Case No. 2, Born in 1929, Male:

This patient was first seen October of 1974. This patient was exposed to mercury fumes for over 15 years, but beginning in 1972 he was exposed to increased quantities of mercury droplets. After three months of this exposure he developed a marked tremor, became irrational and confused, indecisive, and very tired. He described himself as being almost in a stupor. He was then admitted to hospital and given BAL for six weeks. He excreted 168 mg of mercury. May, 1971, he had 115 meg per liter of blood. After discharge he was no longer exposed to mercury, but remained tired and depressed for which he was given an antidepressant. This helped a little, but his personality was now altogether different. Normally, according to his wife, he was easy-going, friendly, outgoing, and cheerful. Now he became irritable and introverted. His wife stated living with him was like living on a powder keg. Also his sex drive disappeared.

He complained that he was subject to excessive daydreaming, felt unreal. He blocked, his mind was in a fog, his memory became poor, and he could not concentrate. His depression had lifted, but he remained very tired.

I diagnosed him as an anxiety state due to residual mercury intoxication. He was started on nicotinic acid, 1 g, t.i.d., ascorbic acid, 1 g, t.i.d., thiamine, 250 mg, one a day, and penicillamine, 250 mg, b.i.d.

By June of 1975 he was much improved. Only the persistent roaring in his ears still remained. He was less confused, more confident, and his potency was normal. No more follow-up interviews were required, and he has not sought any additional help. His HOD scores are shown on top of page 173.

Case No. 3, Born 1936, Male:

This patient was first seen January, 1975. He began his exposure to mercury in 1958. In 1965 he developed what he described as weird symptoms. He be-

	Total	Perceptual	Paranoid	Depression	Schizophrenia
October, 1974	99	21	2	16	4
June, 1975	42	11	1	7	3
Normal scores	0-30	0-3	0-3	0-3	0-1

Short Form for

came nervous, irritable, developed hysterical attacks, and later became depressed. He was diagnosed as having an anxiety state. In 1967 blood mercury levels were very high. At that time the whole department which was exposed to mercury became hyperirritable and were considered screwballs by other workers. The more experienced workers began to avoid mercury and sought help from the corporation and their union. Both ignored their complaints.

When seen in January of 1975 this patient was still very depressed even though he had not been exposed to mercury for a long time. An examination of the mental state revealed the following changes:

Perception —He felt people were watching him, had visual illusions seeing stars, geometric patterns, and his own face altered as he looked at it in the mirror. He heard a ringing in his ears, heard his own thoughts, and heard the thoughts of others and felt very unreal.

Thinking—He was paranoid, delusional about his union and employer, very jealous about his wife. There was severe blocking, his memory was erratic, and his concentration was very poor.

Mood —He was very depressed, irritable, and fatigued, but not suicidal.

Behavior—At times his behavior had been irrational.

I diagnosed him as suffering from a schizophrenic syndrome due to mercury intoxication. He was started on nicotinic acid, 1 g, t.i.d., ascorbic acid, 1 g, t.i.d., thiamine, 250 mg per day, penicillamine, 250 mg, b.i.d., zinc sulphate 10 percent and manganese sulfate 1/2 percent, 15 drops, b.i.d., and Diazepam, 5 mg, p.r.n.

Mercury urine values in January, 1971, were 25 mcg per liter and in June, 1972, 10 mcg per liter.

By February of 1975 he was less irritable and more relaxed. Three months later improvement was steady but slow and nicotinic acid was increased to 2 g, t.i.d. When seen last in August, 1975, he was much improved to the point where he no longer needed to be seen. He had much more energy, no longer had severe outbursts of anger. I considered him nearly well.

His HOD scores were:

	Total	Perceptual	Paranoid	Depression	Schizophrenia
Retroactive for			12	8	12
1970	109	35	8	12	6
January 9, 1975	102	14	5	12	4
February 28	56	11	4	11	5
May 28	55	11	4	4	3
August 29	33	5	0-3	0-3	0-1
Normal Range	0-30	0-3			

Discussion

Many more patients will have to be treated before any firm conclusion is possible. Since I have seen only these three cases and all responded, it is likely that in other series a proportion will also respond, depending upon the degree of intoxication, amount of neurological damage, and so on. Treatment should provide the following:

(a) for restoration of cerebral psychiatric function—vitamin B3 (niacin or niacinamide) and Pyridoxine are chiefly involved here;

(b) to restore cerebral neurological function—thiamine is indicated for this by analogy with its use in the Korsakoff-Wernicke syndrome;

(c) a heavy metal chelator, e.g., penicillamine, to bind and excrete the heavy metal;

(d) extra zinc to replace what the chelator may remove;

(e) ascorbic acid which is a chelator for heavy metals and has been shown to alleviate toxic symptoms produced by mercury and lead;

(f) finally, the diet must be as nutritious as possible which to me means a sucrose-free diet.

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MEGAVITAMIN-INDUCED REMISSION OF ONE CASE OF HUNTINGTON'S CHOREA

Introduction

Huntington's Chorea is a rare (5 per 100,000) degenerative neurological disease first described in 1841, although before that it was known by the local population as "the magrums." George Huntington in his now famous lecture, February 15, 1872, before the Meigs and Mason Academy, Middleport, Ohio, appeared to have described it as an afterthought. He considered it a form of Sydenham's Chorea with a tendency to insanity and suicide, its appearance in adults, and its dominant heredity. He did not consider it important stating, "I have drawn your attention to this form of Chorea, gentlemen, not that I considered it of any great practical importance to you, but merely as a medical curiosity." This information and the clinical description which follows is from an excellent description by Bruyn (1968).

It has been traced back many centuries, but there are no accounts of it before 1841. Inheritance is due to a monohybrid autosomal gene with complete penetrance. Monozygotic twins concordant for Huntington's Chorea have been described. It is found in every country, but is more frequent among lower socioeconomic classes. These families also have a high incidence of neurological disorders, alcoholism, mental deficiency, and insanity.

It usually begins between ages 20 and 50 with a mean age of onset between 35 and 44. Only 10 percent survive beyond 20 years, 70 percent die within 15 years. Its course is "inexorably progressive." However Bruyn mentioned one woman age 87 who developed symptoms at age 14 (out of 120 cases).

Clinically the disease is characterized by involuntary movements involving many muscles and muscle groups. Movements are slow and possess a waxing and waning property which eventually may become athetoid. Each patient has a unique set of movements. Movement becomes awkward. At first patients appear restless and fidgety with occasional grimaces which may be mistaken for tics. Partial opening of the mouth and spasmodic working of the tongue and throat muscles become more

apparent especially when dysarthria develops. The voice loses much of its tonal force. Speech which is affected early in the illness becomes dysarthric, explosive, hesitant, and incoherent, spaced by long silences. Swallowing and respiration become difficult.

Eye muscles are affected. When looking laterally there are saccadic movements. Vertical gaze may become impossible. The head nearly always is in anteflexion, the chin resting on the sternum.

Later choreatic movements become slower and more stereotyped. Muscles of the trunk are involved. Gait becomes uncertain and unsteady. The length of the steps is irregular, and turning presents major difficulty. Muscle force remains unimpaired, but sustained contraction is broken by sudden relaxation. Fatigue is very common.

Psychiatric symptoms nearly always are present and come on long before there are somatic changes. The patients develop changes in perception, thought, mood, and behavior. Perceptual changes are rare. Thought disorder develops. Delusions of grandeur are common, or may take any paranoid form. Concentration is not impaired, and confusional states or delirium are not seen. Mood changes are frequent. The knowledge that one may develop and later does have this disease produces anxiety, and depression with suicide is not uncommon. Patients later are quick and ill-tempered, irascible, moody, and obstinate. Very late in the illness, with progressive dementia the patient may develop euphoria. Behavior is affected, and 20 percent of all patients have been punished for criminal behavior before the disease was diagnosed.

When psychosis develops early in the illness, the first three years, it tends to be schizophrenic in form. A second peak incidence occurs during the middle third of the illness and is then more like an organic psychosis.

According to Bruyn (1968) the rather scanty research into the biochemical lesions had not yielded any significant
leads. Perry et al. (1973) compared autopsied brains of patients with chorea against control subjects. There were two major differences.

The choreac brains contained much lower quantities of gamma amino butyric acid (GABA) and homocarnosine, a related substance in the substantia nigra, caudate nucleus, and putamen-globus pallidus while glycerophosphoethanolamine concentrations were elevated. These authors suggested that deficiency of GABA, a possible inhibitor of synaptic transmission in the brain, might be responsible for the symptoms. This could result from reduced activity of the enzyme glutamic acid decarboxylase.

Bird et al. (1973) measured the activity of glutamic acid decarboxylase (GAD) in postmortem brains of 14 cases. Its activity was reduced by 80-90 percent in the basal ganglia. Stahl and Swanson (1974) also found a marked reduction in GAD activity.

The only definite finding, therefore, is a reduction in GAD, the enzyme which converts glutamic acid to gamma amino butyric acid, and a decrease in GABA. Another interesting finding is that these changes occur in the darkly pigmented areas of the brain, the substantia nigra, caudate nucleus. In these areas the pigment is believed to arise from noradrenalin and adrenalin to form quinoid melanins (from noradrenochrome and adrenochrome, Hoffer and Osmond, 1967). In albinos who lack the enzyme necessary to oxidize dopa to dopachrome these areas of the brain retain their normal color. Finally, it has been known for many years that adrenochrome is one of the most powerful inhibitors of glutamic acid decarboxylase.

Because Huntington's Chorea is so rare it is very difficult to gather a series. In nearly 25 years of practice as a psychiatrist I have seen only one case. This one case is the subject of this report. After 20 years of gradual but progressive deterioration, this process was halted and reversed by megavitamin therapy. Since so far no one has reported such a similar

event, I believe it is important that it be recorded so that other physicians who run across cases of this dreadful disease will attempt a similar therapeutic regime. It is important to determine whether my case is the only one who will respond in this way, whether he represents a subgroup of Huntington's Chorea, or whether a major portion may be expected to respond.

Case History

Mr. A., born February, 1913, consulted me October, 1973, complaining of nervousness present since he was 40, but much more severe over the previous year. He had no appetite, was weak and tired, and had gone down in weight from 165 pounds when he was 40 to his weight of 130 pounds. He had been aware of marked muscle wasting which was generalized. He was so weak that his main preoccupation had to do with the routine necessities of survival, eating, dressing, and so on.

With his wife they had become very interested in nutrition, and they had both gone onto a sugar-free, high-protein diet. He had quit smoking one year before and felt that this had been helpful. Five years before he had consulted a psychiatrist who, he stated, had not been helpful.

When I examined him there were no perceptual changes, his memory for recent events was poor, his concentration had deteriorated, and his thinking was interrupted by pauses (blocking). In his mood he was depressed, nervous, tense, very irritable, and occasionally too angry at others. He suffered from a peculiar obvious muscle twitch even when asleep. Occasionally he suffered from muscle cramps in his legs and stumbled a lot when he walked.

Family History

Father—1876-1944: This man was re-admitted to mental hospital in 1932. He was thin, had many purposeless movements of arms, legs, body, and head, and was very restless. He was depressed and blamed himself. He was also delusional, believing he was getting messages from God. At times he had marked choreiform movements.

He was first committed to the mental hospital in 1930 and diagnosed as involutional melancholia. After discharge he was slightly improved.

During his prolonged stay in hospital he occasionally suffered from hallucinations. He was reluctant to discuss these. He died of cerebral hemorrhage.

His father had been mentally unstable for a number of years, and one brother was similar to the patient for about three years when he died. One sister was normal.

Siblings: Mrs. A. brought in the following account of the five brothers:

(a) Born 1908—As a boy he was very shy and showed extreme embarrassment. He carried his head tipped to one side, had noticeable hand gestures, and **a** loose movement of his legs. These gradually worsened as he matured. However, he was intelligent, a good student, and served in the RCMP until failing health forced an honorable dis charge when 38. Two years later he became quarrelsome, irritable, corn-plaintive, withdrawn, and lost self-confidence. He became paranoid, always blaming others. He lost weight rapidly, becoming emaciated. He entered a convalescent home at age 64. Eventually he could no longer walk and speech became unintelligible. In 1973 he was slightly better and was able to sit in a recliner and able to say a few words.

(b) Born 1909—Normal.

(c) Born 1911 —Normal.

(d) Born 1913—Mr. A.'s wife des cribed him as being very embarrassed as a teenager and very nervous. He was very sensitive to pain and burns. In public his hands developed a tremor, but he was a good student. When he reached 40 his personality changed from a quiet person ality and he became overbearing and adamant, withdrawn, and lacked con fidence. By age 55 he was no longer able to work enough to earn a living. By age 57 he could not work at all. He was very

thin, and his muscles were wasted. He angered easily and became irresponsible.

(e) Born 1915—Between 20 and 30 he became alcoholic and his personality deteriorated. At age 35 his movements became awkward and he could not walk properly. At age 38 he joined AA. He now suffered severe incoordination and often appeared drunk, but had not been drinking. At age 50 he was placed in a convalescent home.

In 1973 he was badly deteriorated and unable to look after himself. He was unable to speak at all, but appeared to understand when he was spoken to. He died in December, 1974.

Children —There are none.

Diagnosis—Anxiety state—Huntington's Chorea.

Treatment and Progress

(a) Dietary—He was advised not to eat any food to which sugar had been added and to avoid all processed and refined foods.

(b) Vitamins—ascorbic acid 1 g, t.i.d., megavit, t.i.d. (each tablet contains 100 mg of thiamine, 25 mg of riboflavin, 100 mg of Pyridoxine, 200 mg of niacinamide, and 500 mg of ascorbic acid. Vitamin B12, 1 mg per week. Weight 130 pounds.

One month —There was a sudden improvement to a flatter plateau of health where he remained. He felt much less depressed, was stronger physically, and had fewer muscle cramps. His concentration was better. He had been able to work outside on his roof. This had been the first useful work he had done in many years. Niacin, 1 g, t.i.d., and folic acid, 5 mg, b.i.d., were added plus magnesium sulfate in solution to provide magnesium.

Two months—He reported he stumbled less often, only occasionally had cramps, but noted no other change. He was free of depression and stated if he had to stay at his present level of improvement the rest of his life he would be content. The niacin was doubled to 2 g, t.i.d., and he was started on vitamin E, 400 units, b.i.d.

Four months—There was no additional improvement, but he had more insight and was concerned about his outbursts of anger against his wife. He was now bored with nothing to do. When first seen he was so busy staying alive he had no time to be bored. All natural functions were normal. He began to consider going back to work.

Six months—There was no change except his weight had dropped to 125 pounds. Because of his excessive fondness for dairy products and a prior history of severe sinusitis I advised him to try a two-week dairy-free program. **Seven months—** There was no improvement on dairy-free and he resumed consumption of dairy products. He had not gained any weight and was becoming weaker and tired more easily. I now concluded that the whole megavitamin program had failed even though he felt much better, as he had lost five pounds from the time I had first seen him (about one pound per five weeks). The objective evidence of weight loss overpowered the subjective assessment of improvement. At this time I increased the vitamin E to 800 units, b.i.d.

Eight months—His weight was about the same.

Nine months—Weight went up to 127 pounds.

Eleven months—Weight was 135 pounds. His appetite was good. His wife reported his muscles were regaining their size, tone, and power. He remained normal mentally. In order to study the effect of more vitamin E it was doubled to 3,200 units per day. The niacin was reduced to 1/2 g, t.i.d. He had normal energy and his chest which had been drawn in regained its normal size. All muscle tremor and cramps were gone. They both felt the major change came when the vitamin E had been doubled from 800 to 1,600 units per day.

Thirteen months —His weight continued to increase reaching 139 pounds. He became so active physically it settled down between 135 and 136 pounds. Niacin was replaced by niacinamide 1/2 g, t.i.d., because he felt niacin caused his nose to swell. There was still a slight

unsteadiness when walking when he was excited.

Fourteen months—He continued to improve steadily and no longer had outbursts of anger.

Fifteen months—He had suffered a cold and lost several pounds which he regained rapidly. Before the cold he had been able to shovel snow. One year ago he had not been able to do any work for longer than 10 minutes. He was again started on niacin, 1/2 g, t.i.d.

Seventeen months —He and his wife reported he was now as well as he had been before the onset of his illness at age 40. Niacinamide was increased to 1 g, t.i.d., to try and improve weight gain which still remained around 135.

Nineteen months—He was taken off niacin to see if he still required it. (He was still getting 600 mg per day of niacinamide).

Twenty months—Normal but slightly more irritable. He was given Diazepam, 5 mg, p.r.n.

Twenty-two months—He reported that about two weeks after he discontinued niacin he became very restless and nervous and would tighten up his body frequently. He resumed niacin medication, 1/2 g, t.i.d., and within a few days he was much better, but he lost three pounds reaching 132 for the first time in a year. When he walked his legs became stiff.

Discussion

When this patient first approached me I could offer no treatment. I was convinced then that this was a progressive deteriorating disease for which there was no treatment and from which no one recovered. The patient and his wife were very knowledgeable about the disease and expected no cure. They had sought me out because they had heard of my association with the use of megavitamins. They had on their own started on a regime for improving their nutrition; they merely wanted my advice on the best possible combination of vitamins. I advised them that the most that they could expect would be a strengthening of his resistance against the ravages of the illness.

I started them on the megavitamin program for the following reasons:

Vitamin E—Because of a clear relationship between muscular dystrophy in animals and lack of vitamin E, and because of the severe loss of weight and loss of muscle mass in Huntington's Chorea and muscular dystrophy, it seemed appropriate to consider this vitamin in megadoses.

Vitamin B3 — A large number of studies have shown that large doses of this vitamin when combined with other components of an Orthomolecular program substantially improve the outcome (Hawkins and Pauling, 1973). I have been using it since 1952 in double-blind, single-blind, and clinical studies, and have concluded it is therapeutic. Since my patient was beginning to show psychiatric symptoms it appeared to me wise to also give him this vitamin. His relapse over one month when it was discontinued proved this was a good decision.

Other B vitamins—These were added merely to balance up the water-soluble vitamin program.

Ascorbic Acid—This was also added to balance up the vitamin program and also to decrease the tendency for infection and colds. My observation on over 20 years of prescribing ascorbic acid have convinced me of its value (Stone, 1972).

Polypharmacy

It is passing strange how the meaning of words becomes corrupted. Polyphar-makos originally meant a physician skillful in the use of drugs. By many it has become corrupted to mean that too many drugs are being used on one patient. The use of a large number of vitamins may appear to be the latter sort of activity, but if we get away from the single nutrient - single disease idea so common among nutritionists and if it is remembered that there is no treatment, then it is rational to use everything which might conceivably help the patient. The clinician is interested in the welfare of his

patient. It would be preferable to know exactly which chemicals are important. There are two ways this can be done: (a) By starting with one nutrient at a time, allow it to work for a reasonable period of time, then to try another one. On one case double blind is impossible, but with Huntington's Chorea not necessary since there have been no recoveries ever published. The double-blind approach would satisfy the purist who sees no problem in waiting decades, but it would not satisfy the patient whose life expectancy is measured in years, not decades. (b) By starting with what might be helpful. If there is no response, then one can conclude none of the nutrients are of value. If there is a response it shows that one or a combination are effective. Once the patient has been improved one can at one's leisure withdraw one at a time and hopefully will be left with the ones which are effective.

It now appears from my study of this case so far that at least vitamin E and nicotinic acid in megadoses are essential. On the comprehensive program given him for seven months including vitamin E, 800 units per day, there was a general improvement but his weight continued to deteriorate, an ominous sign. He was beginning to get weaker as well. The month after the vitamin E was doubled to 1,600 units there was a major improvement, and for the first time in many years the steady drift downward in weight was reversed and he gained. He gained eight pounds in two months. This gain continued until he reached 139 pounds when his increased physical exertion and ability to be more active settled it down at around 135 pounds. When the nicotinic acid was eliminated he began to relapse within two weeks with a marked increase in restlessness and nervousness. He promptly went back on, but his weight had dropped rapidly to 132 pounds, the largest drop over a similar interval since he had become my patient. These changes in relation to his medication therefore suggest that both vitamin B3 and vitamin E are important. Many years ago my colleagues and I suggested that adrenochrome was involved in the etiology of schizophrenia. The arguments pro and con are discussed in Hoffer and Osmond (1967). It is possible that adrenochrome (or nor-adrenochrome) is also involved in Huntington's Chorea. This is suggested by the following evidence:

1. Adrenochrome is a powerful inhibitor of glutamic acid decarboxylase. This would lead to a decrease in GAD levels and in GABA levels, as has been found by Perry et al. (1973) and Bird et al. (1973).
2. This occurs primarily in the pigmented basal areas of the brain where the melanin is probably of noradrenalin or adrenalin origin, not from tyrosine.
3. The psychosis of Huntington's Chorea has a schizophrenic quality in its early stages.

If this hypothesis is correct, one can account for the therapeutic action of the vitamins as follows:

Vitamin E—By reducing peroxidation and decreasing formation of the chromes derived from the sympathomimetic amines.

Nicotinic Acid—Increasing nicotinamide adenine dinucleotide levels would decrease the oxidation of these amines (see Hoffer and Osmond, 1967). **Pyridoxine—**Increasing conversion of tryptophan into NAD would have a similar effect.

Ascorbic Acid—By inhibiting oxidation of the amines.

Vitamin E has been described as a vitamin in search for an indication (Tappel, 1973). This is the general opinion of most nutritionists. However, many clinicians are convinced that many indications exist already. They point to a huge number of clinical reports which suggest that a few conditions do respond rather well to vitamin E therapy. Unfortunately many of the clinical studies were developed before the so-called double-blind controlled experiment became popular. Since then it is a general belief among most medical investigators until it has been hallowed by the double blind with a significant difference over

the 5 percent level of probability. The fact is that no one has yet run that crucial experiment to determine that the double-blind methodology does what it is supposed to do theoretically. It has never been established by experiment. It is like using a method for measuring cholesterol levels which has never been standardized against any previous method. Because few of the vitamin E studies have been double blind, few physicians have been interested as a class. A large number do use it for their own families and for their patients. If we define as a megadose a level of 800 I.U. (mg) per day or more, then we find that all studies can be divided into two classes: (a) those under 800 mg, usually under 400—most of these studies have been negative; (b) those over 800 I.U. —most of these have yielded positive results.

I have been interested in the relationship of vitamin E to muscle metabolism. It is now established that vitamin E deficiency will produce muscular dystrophy with stiffness, lameness, and heart failure in a variety of farm animals, but also in the monkey (see Roche, Vitamin E for Farm Animals). The deficiency is worse if there is also a deficiency of selenium, or with an excess of polyunsaturated fats. Roche concluded, "It can no longer be assumed that natural foodstuffs will always provide physiologically optimal levels in rations," especially since the turnover of vitamin E is rapid and it cannot be stored like vitamin A.

Human muscular dystrophy has not responded to smaller doses of vitamin E, but this should not be surprising. Anderson (1973) pointed out that short-term trials with antioxidant supplementation would not show any significant reduction in the incidence of myocardial infarction. Once lipid peroxides have been incorporated into tissues their removal is slow and difficult. This would also apply to dystrophic muscle. In this report Anderson concluded that a combination of increased dietary unsaturated fatty acids plus inadequate intake of vitamin E would lead to the formation of highly reactive lipid peroxides in both skeletal muscle and in the myocardium.

Vitamin E has been recommended for treatment of certain myocardial diseases (Shute and Taub, 1969), for prevention and treatment of leg cramps (nocturnal leg cramps restless legs syndrome, Cathcart, 1972; Ayres, 1972), for management of arteriosclerotic occlusive vascular disease (Toone, 1967), for intermittent claudication (Williams et al., 1971), and wheat germ oil helped 13 out of 107 patients with a variety of neuromuscular disorders (Rabinovitch et al., 1951), some of them dramatically.

There is thus evidence linking vitamin E with muscular dystrophy. In order to establish a firm relationship a long-term study will be required using megadoses of vitamin E. It will have to be combined with an optimal diet and fortified with vitamin A and vitamin D3 to balance out the intake of vitamin E.

The old idea of one vitamin-one disease is- no longer correct, useful, or adequate.

So far no other program has been successful although modern therapeutic agents have been helpful in controlling one or another facet of the illness. For this reason I would suggest that other cases with Huntington's Chorea be started on a similar broad-spectrum, megavitamin nutritional approach.

It would then be possible to determine how many would respond, not just by some slight improvement, but by restoration of normal function. If more patients do respond, it might be possible to hypothesize that Huntington's Chorea is a vitamin E-dependency disease coming on during adulthood, perhaps as a result of a chronic vitamin E deficiency.

But due attention must also be given to the possible role of food allergies.

As a rule one case is not convincing except in a hopelessly deteriorating disease like Huntington's Chorea where it becomes as important as one white crow in a flock of many blacks. It brings attention to the possibility of a new treatment approach. Two cases would be

even more impressive. There is such a case which came to my attention. The information was given to me by the patient's husband. I have not examined her as she does not live in North America. On April 17, 1975, her husband reported that his wife, age 32, had first suffered symptoms at 19, slurred speech, lack of facial expression. Her father died psychotic in a mental hospital about one year after admission. The symptoms came on after the birth of her second child. They were primarily psychiatric-frequent episodes of depression and excitability. They both became aware that these outbursts tended to come after certain meals and stresses such as cold, fatigue, or too long an interval between eating. They began to eliminate certain foods, especially meats, and her furious outbursts gradually subsided. At age 22 she developed fearful nightmares and a nervous tic and involuntary winking. At 24 she was diagnosed as Huntington's Chorea and advised there was no treatment and to prepare for her inevitable deterioration and death. At 24 her movements were very jerky and she developed a pin-rolling movement of her hands. She was again diagnosed at the neurological unit of a large hospital and started on Librium and Tetrabenazine. The only change was an improvement in tremor.

By now she weighed 238 pounds (57") due to her excessive craving for sweets and her habit of eating up to eight meals per day. Suddenly her weight began to drop and by age 28 she was 140 pounds and still dropping. She was also weaker, her muscles were thin, soft, and wasted. When she was 29 (three years ago) her husband read the book by Shute and Taub (1969) on vitamin E where Huntington's Chorea was not discussed. But he reasoned that it might improve the micro circulation and thus improve oxygenation. He started her on vitamin E, 200 I.U., once a day. Very soon she was brighter, more energetic, so he doubled it to 400 I.U. The results were astonishing; whereas before she had to be in bed by 8 p.m. she could now go to parties and stay awake until 5 a.m. After increasing the dose to 600 I.U. he noted her muscles were firmer. Her weight stabilized at 126 pounds where it is today. She. was now on a vegetarian diet fortified with other B vitamins and vitamin B12. All her gastrointestinal complaints such as constipation, etc., cleared. She was more relaxed and had few outbursts. She remained nearly well except she continued to have difficulty in speech, had difficulty doing simple tasks, required more rest. Early in 1975 she was examined because of a sore throat and her husband was given a very poor prognosis. At the beginning of June the vitamin E was increased to 1,600 I.U. There was a prompt improvement and she can now go on walks which she had been too tired to do before. When this report was prepared (October, 1975) she was still improving.

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