Case Presentations of Orthomolecular Disturbances in a Family Practice

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I would like to present two different types of Orthomolecular disturbances — one very rare and genetically produced and the other much more common and iatrogenically produced.

During April, 1973, an intelligent mother visited my office on referral from Dr. A. Hoffer. She related the history of her 12-year-old female child, while the child waited in the outer office. The birth weight of the child was 5 lb. 8 oz. The infant failed to thrive, lost weight, and was jaundiced. A total blood replacement was performed but the child did not improve. After three weeks she went to a University Hospital in a larger city, where eventually the diagnosis of galactosemia was made. A Molls diet was ordered. The child was referred back to a pediatrician in Calgary who invested a great deal of time and effort in locating vitamins which were lactose-free, to use for this child. She remains retarded, attends a special school, and will probably never go beyond the grade four level in the special school.

Three more children followed, a boy age 11 with galactosemia, a girl age 10 who is normal, and a girl age eight with galactosemia.

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The 10-year-old girl is very bright and is at the top of her class. The 11 and eight-year-old children were immediately diagnosed at birth by the pediatrician as having galactosemia. They were placed on a lactose-free diet and lactose-free vitamins. These two children are described as being a bit slow, but not retarded. The 12-year-old girl is tall and thin, has wiry hair, dry skin, and spindly fingers. I spoke with the pediatrician who had not seen the children for seven years and he agreed with me that genetic investigation and counselling was certainly indicated at this time, since this had not been done before. This will now entail the investigation of at least 12 people, since the mother's brother is married to the father's sister, and this marriage has produced four normal children. I have given the responsibility for this investigation to the Director of Pediatrics, Dr. R. Blake at the Calgary General Hospital. This investigation can partly be carried out in Calgary, and the remainder by sending specimens to Duke University or elsewhere. My part in the care of the three children is to prescribe lactose-free megavitamins, and make sure that the parents' hopes are not unduly raised. Sugar and starch-free vitamins are available in Canada and the United States.

Galactosemia is an inborn error of galactose

metabolism due to the congenital deficiency of the enzyme 1-phosphate uridyl transferase, resulting in a tissue accumulation of galactose 1-phosphate.

Certainly no blame can be attached for missing the diagnosis of galactosemia originally, since, in a survey in the New England states, one case occurs in anywhere from 180,000 to 340,000 deliveries.

In a similar vein I have recently been treating two middle-aged ladies, both diagnosed as having hypoglycemia. In one, the blood sugar level at three hours dropped to 27 mg percent. Both ladies on a hypoglycemia diet only improved minimally. Both had gastrointestinal and multiple psychosomatic-type complaints. One, age 41, was completely investigated by a gastro-enterologist and nothing was found except a persistently elevated sedimentation rate of 40-50 per hour. In desperation and with the patient's request, I referred her to a second gastroenterologist. He investigated her in hospital and diagnosed a lactose intolerance. A lactose-tolerance test produced a subnormal rise in blood sugar, with urines positive for lactose at two and three hours. She is now quite well on a lactose-free diet. Occasionally she has the craving to eat cottage cheese or cottage cheese pancakes which she loves. When she yields to this craving, she promptly develops bloating and diarrhea.

The other lady, age 49, made the diagnosis herself. She recalled that as a child she could not tolerate milk. She stopped consuming all dairy products, and combined this with a hypoglycemia diet, along with injectable vitamins. She fairly quickly became well. After one month she thought that, as a test, she would eat some yogurt. She became quite ill. I am sure that she will not test herself in this manner again.

While glucose intolerance is fairly common, lactose intolerance is less common. I believe that when a patient with hypoglycemia doesn't do well on a hypoglycemia diet, it certainly would be worth-while testing for lactose and other food intolerance.

Dealing with the more common and iatrogenically-produced Orthomolecular disturbances, during the past four years I have treated 12 patients who have had a Billroth II surgical procedure for peptic ulcer disease and have suffered severe orthomolecular-type complications following surgery. Ten of the 12 patients were males.

Some years ago this procedure, which consists of a high gastric resection and a gastroejunostomy, was a common initial surgical procedure for peptic ulcer disease as well as for cancer of the stomach. Because of the high incidence of serious complications, including dumping syndrome, malabsorption and vitamin deficiency, maldigestion, cerebral, mental, and psychosomatic-type complaints, this procedure has mainly been discarded. It is now performed in cases of cancer of the stomach and only as a last resort procedure in peptic ulcer disease. We must all accept the responsibility for this iatrogenically-produced disease, the referring physician as well as the surgeon. This is not particularly cited as criticism, since with hindsight everyone can recognize the deficiencies of the procedure. To a lesser degree, the dumping syndrome may also complicate a Billroth I procedure as well as pyloroplasty and vagotomy. The Billroth I procedure is a lower gastric resection and gastro-duodenostomy, and as such is more physiological than a Billroth II procedure. Pyloroplasty consists of simply widening the lower end of the pylorus and first part of the duodenum, and as such I consider this to be the best initial surgical procedure for peptic ulcer disease.

There is a vast amount of literature on this subject. I would like to quote from Dr. Leon Frankel (1971), St. Luke's and Children's Medical Centre, Philadelphia: "Pragmatic conversion from a Billroth II to a Billroth I procedure or vice-versa, for the correction of dumping syndrome, does not establish the principle of physiological — anatomic

relationships. A means for abolishing the iatrogenic factors is in the surgeon's hand once again." This does refer to surgical procedures advocated for the correction of the dumping syndrome. I do not fully agree with this concept. I feel that surgery should be a last resort, only after medical management has failed. In many of these cases Orthomolecular methods will succeed after a biochemical investigation has been completed.

In order to resist the temptation of selecting the most successfully treated of the 12 patients, I will only present the first patient investigated and treated, and my most recent patient. I might add that none were medical failures, no two cases were alike, and none required further surgery.

CASE 1 is that of a married male. He was the manager-owner of a small busy hotel. He gave a long-standing history of a chronic prepyloric ulcer. In October, 1967, he had a Billroth II procedure performed. Following discharge, he next spent eight days in hospital during November, 1967, complaining of listlessness, indifference, vertigo, fatigue, and anorexia. He was found to be negativistic. The family physician made three tentative diagnoses: (1) postoperative delayed psychosis, (2) catatonic schizophrenia, and (3) hysteria. The discharge diagnosis was acute anxiety. His next admission was in January, 1968, for 33 days. On this occasion he was treated by a family physician, an internist, and a psychiatrist. His final diagnosis was: (1) anxiety depression and (2) overdose Tuinal®.

Since his surgery he was depressed, he had paranoid thinking, and he was constantly receiving antidepressants. He was treated with a course of seven ECTs.

His next admission was for three days only during December, 1968. He had previously been working very hard and there was some exposure to and inhalation of smoke in fighting a fire near his hotel. On admission his BP was 70/50 and temperature was 104 degrees. A chest x-ray revealed "an increased density of the right lung base." An EEC read in part "diffuse cerebral process as intoxication. Viral or other metabolic process cannot be ruled out." The final diagnosis by the family physician was **"Exhaustion Syndrome."** The psychiatrist's diagnosis was **"severe neurotic with hysterical reaction."**

Ten days later, on January 2, 1969, he presented in my office for the first time. He was still obviously quite ill. He was nervous, depressed, fatigued, perspired freely, and his thinking was disturbed. He had never felt this way prior to gastric surgery. He was a non-drinker and a non-smoker, and there was nothing relevant in his past history other than chronic peptic ulcer disease. A HOD test (Hoffer and Osmond, 1961; Kelm et al. 1967) done at this time revealed a depression score of 13, a perceptual score of 21, a paranoid score of 7, and total score of 103, all markedly elevated. I made a tentative diagnosis of schizophrenia but did not inform the patient of my diagnosis. I prescribed nicotinamide and vitamin C — 3 gm of each daily, Mellaril 75 mg daily, and Surmontil 75 mg daily. Over the next two weeks he improved only slightly and then spent two weeks with his wife on vacation in Hawaii.

On return from vacation his wife stated that he vegetated while they were away and I began to suspect a dementia. A referral to a neurologist was made. His EGG was still abnormal and the neurologist made a tentative diagnosis of dumping syndrome. He was admitted by me to hospital from April 15-25, 1969, and at this time the most significant finding was that of a three-hour glucose level of 30 during a six-hour glucose-tolerance test. He was placed on a high-protein, low-carbohydrate diet with frequent feedings and his vitamins were increased to include Allbee with C twice daily. He was improved on discharge. During his illness he was unable to work and his wife mainly covered for him.

Following discharge from hospital I saw him briefly about every three months. After

remaining well for almost three years, he decided to stop his medications and he neglected his diet. Within about one month he had a most severe relapse of his illness and spent about six weeks in hospital under my care. A repeat EEG at this time was normal. I then administered a course of eight unilateral ECT, restarted his megavitamins, and placed him back on his diet. He was discharged on May 26,1972, well. He has remained well since and takes no psychotropic drugs. I now see him only once a year and last examined him on May 1, 1973. His HOD test at this time revealed a depression score of 2, a perceptual score of 2, a paranoid score of 2, and a total score of 15 — all within normal limits. I think it was superfluous for me to remind him to remain on his vitamins and diet for the rest of his life. He has worked steadily during the past year and is presently negotiating the purchase of another hotel.

A brother of this man, who is two years older, has been a patient of mine for 16 years. He had a chronic gastric and duodenal ulcer history of 25 years' duration. On December 21, 1972, he had a Billroth I procedure and a vagotomy. He has been well since and has gained 23 pounds.

CASE 2 is that of a 71-year-old divorced man who first visited my office on November 21, 1972. He looked much older than his stated age and my first impression was that he was suffering from an advanced terminal cancer. Much later I reviewed his hospital record. He had a Billroth II procedure in 1963. He had a history of gastric distress for about one year prior to this surgery. Even at this time there was a strong suspicion of malignancy. Prior to surgery he had an esophagoscopy and biopsy of the lower end of the esophagus which was negative for malignancy. His Hgb. was 7.3 gm and E.S.R. was 65. A preop diagnosis of linitis plastica was made. However, again the pathological report revealed no evidence of malignancy and read in part "mild atrophic gastritis."

He was not improved by surgery and over the next 10 years he had been treated by many physicians. His last physician was an internist who had attempted to help him over a five-year period. He was finally advised to find another doctor.

When I first saw him he was anxious and depressed, he complained of bloating, anorexia, and the inability to digest meat. On sitting down to eat a hot meal, a flush would come over him and he became nauseated. He had lost weight. In addition to his gastric surgery he had had a cholecystectomy, a prostatectomy, and four major operations on his lower back and neck. A complete physical examination revealed only emaciation and some osteoarthrosis. A laboratory work-up revealed only one very significant finding — that of a serum B12 level of less than 50 uugm per ml (normal for male — 330-790 uugm per ml). A HOD test revealed a depression score of 10, a perceptual score of 9, a paranoid score of 1, and a total score of 40.

I advised hospitalization for a more complete investigation but he resisted this suggestion. By December 1, 1972, he was feeling much worse — he would eat a spoonful of food, develop a hot flush, and was unable to eat any more. One evening he passed out. He was dizzy, unsteady on his feet, had marked weakness, anorexia, and nausea, and complained of generalized pains. For the first time he was willing to enter hospital and was hospitalized for three weeks. Bone marrow studies were normal, a Schilling Test without intrinsic factor was normal, and surprisingly a five-hour glucose-tolerance test was normal. With a complete investigation no evidence was found of malignancy. I began treating him with daily intramuscular injections of 1000 mcg of vitamin B12 and megavitamins by mouth. All his symptoms, except his joint pains, cleared completely. He was discharged on vitamin C and nicotinamide $1^1/2$ gm of each, Pardee 1 daily, Allbee with C 550 T daily, Folic Acid 5 mg TID, a-Tocopherol 800 IU. daily, and B12 injections 1000 mcg monthly. I saw him last on May 1, 1973. He has been gaining weight steadily and feeling well. His only

residual complaint is that of some slight muscle aches and joint discomfort. A HOD test at this time revealed a depression score of 1, a perceptual score of 5, a paranoid score of 0, and a total score of 10, all normal.

For the first time on this visit he informed me that his friends gave him up for dead when he entered hospital and that he himself felt this way, and that this was his initial reason for resisting admission to hospital.

What was most surprising and puzzling about this case was that the patient suffered such devastating illness, and with a most extensive investigation the only real positive finding was that of an extremely low serum B12 level. With megavitamin treatment he made a most satisfactory recovery. I would suspect that this man has suffered from a chronic vitamin B12 deficiency with a multiple-vitamin dependency state for at least 11 years. Certainly the Billroth II procedure did nothing to improve his health.

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