

MULTIPLE SCLEROSIS—AN INTERNISTIC ILLNESS

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Altogether, from July, 1964 to March, 1986, we have treated about 1,600 MS patients in the Paracelsus Hospital at Silbersee, Hannover-Langenhagen, some as inpatients, some as outpatients. About 1,300 of these patients came from North America, the rest scattered from Mid-Europe, Northern Europe, the Mediterranean, South Africa, Australia, Tasmania and East Africa.

At the present time, the full scope of the etiology and mechanism of MS cannot be given—only in a very condensed manner. The primary characteristic of the MS patient is damage to the cell membrane system. This is essentially caused by an insufficient amount of colamine phosphate (2-aminoethanol phosphate or EAP) in the lipid-system of the outer cell membranes. We are indebted to the distinguished biochemist ERWIN CHARGAFF for the discovery of the significant role that colamine phosphate plays in the cell membrane system. (Columbia University, New York) Patients who develop autoimmune diseases later in life, (not only MS) are characterized by a lack of colamine phosphate in the cell membranes. (GALLAND) This imperfection in the construction of the cell membrane explains certain familiar occurrences; among others, a series of disturbances in the function of the membrane: with MS patients, the cell membranes are generally characterized with an unusual porosity, especially however, with a reduction of its ability to act as an electrical condenser.

We know from the research of PRESSMAN (New York), that the colamine phosphate salts integrated in the membranes function both as a neurotransmitter and as a biochemical binding station for minerals—calcium for example. The vitally necessary condenser effect of the cell membranes can be obtained only through this calcium binding in the membranes. This idiopathic membrane deficiency appears to be a "primum movens" for the development of the disease. It has the following consequences:

1. The ability of the cells to completely extinguish certain virus genomes is defective on account of the electrostatic membrane deficiency. COOK and co-workers (New York) have discovered that MS patients and also victims of ALS (amyotrophic lateral sclerosis) are unable to extinguish the residual genomes of measles virus, distemper virus, cattle plague and swine fever in the epithelial cells of the upper small intestinal tract, as they do for other viruses. This phenomena has not been observed in patients which do not have MS or ALS.
2. The endogenous membrane defects previously referred to especially, are an invitation to the development of the auto-immune process. This happens

because of the electrostatic, repelling effect of the cell membranes, and also their lacking proper structural coherence to keep in step. There is substantial evidence that the persistence of virus genomes in the tissue, activates and accelerates this immune process. (Investigated by MANNWEILER from the Pette Institute for 20 years.)

3. Since the myelin takes the form of a multiple winding of the cell membrane system, we are especially concerned with the lessened condenser activity of this function. Up to about three years ago, it was assumed that myelin was an insulation material around the central axon. For this reason, it created quite a sensation when a research group from Buffalo, New York discovered that an electrical shunt exists between the myelin sheath and the central nerve axon. This was discovered three years ago. Now we must consider the myelin system, not as an insulation, but as a Tesla energy system. The principle is that since the nerve cell impulse is much too weak to start a synaptic reaction in the affected organ, a strengthening is necessary. This takes place in this way: the neurogenetic impulse in the membrane system of the myelin is conducted in such a way that a corresponding amount of Tesla function energy is derived from the scalar electromagnetic (tachyon) field from aether space. This raises the neurogenetic energy potential to a two or three times higher amount. (This is the same system that we recently developed for the so-called Plasma Ignition, which allows us to burn gasoline more efficiently in automobiles and so use a leaner mixture.) When the condenser function of the myelin membrane is damaged, the conversion ability and the synaptic stimulation of the organ that it affects will be below par from an energy standpoint as a result of this. As the condenser function of the harmed membrane is further harmed by the autoimmune process, the illness escalates.

There is also a series of supplementary conditions that have an active influence upon the course of the illness. These are:

a) Patients who live or work in geopathogenic zones, which result in an additional membrane discharge. (For illustration of scroll waves, Los Alamos National Laboratory, New Mexico, see page 304 of my book Revolution in Technology, Medicine and Society.)

b) MS is much more prevalent in the sections of the northern and the southern hemispheres where there is a dairy industry and dairy products are marketed. It may be that the viruses in the milk play an additional etiological role, or it may be the glutenes. (Cancer of the breast is also higher in the areas where dairy products are marketed.)

c) MS in the US and Canada shows a considerable variation in symptoms and in the course of treatment from the MS in Europe, South Africa and Australia. The most probable cause for this is the large amount of aluminum foil and aluminum cans used in the food and drink industry plants. It is also true downwind from other plants where the atmosphere contains traces of platinum, nickel, chromium or (and especially) fluorine. (The same pattern is true for ALS.) While the harmful effect of aluminum upon the nervous

system is quite well known, we also know that fluorine and heavy metals can destroy the control capabilities of a series of little known dihydroxy steroids which are able to repair both the derailing of the immunological surveillance system and the genetic surveillance system. (FISSER) (Aluminum is now known to play a role in Alzheimer's disease.)

d) The tendency of MS patients to develop infections in the urinary tract is not only caused by a neurological disturbance of the bladder function but also by damage of the electrostatic defense capabilities against bacteria in the discharging urinary tract, on account of the previously referred to reasons. The same reasons are responsible for a series of additional phenomena such as capillary fragility and chondritis.

The MS patient not only feels cold—he actually is cold. His body does not generate sufficient heat. The origin of this phenomena, apparently lies in the damage to the condensor function of the entire body cell membrane system, resulting in a lessening of the heat production, generated from the scalar electromagnetic field. About 4° C of body temperature is generated from this energy rather than from nourishment in adults—the percentage is higher in children. The most important part of MS therapy is the repair of the condensor function of the cell membranes. This defect occurs at the bonding station for minerals—and the best way to correct it is to use colamine phosphate salts to transport the needed mineral in. The only satisfactory salts are calcium EAP, magnesium EAP, and potassium EAP. The relevant preparations are phosetamin (enteric coated tablet), calcium EAP (enteric coated tablet) and calcium EAP in solution for IV. In fact, the last mentioned preparation was officially declared to be a MS remedy in 1967. When there is also a necessity of producing an immunosuppressant therapy—a question which we can answer after a test for the consumption of naked-nuclear lymphocytes—we use trophosphamide (Ixoten). We stopped using immunosuppressive therapy with Azathiaprine in 1968 because of possible side effects on the liver and bone marrow.

In case of infection of the urinary tract, we usually use Harnesal, since it has been known for 25 years (from the research of Hackmann-Bayer) that this sulfonamide appears to be adapted for unlimited use, not on account of its bacteriostatic activity, but especially for its electrostatic surface properties. Schedule for colamine phosphate therapy:

7 tablets of phosetamin^{*} daily—to insure good flow of bile, which is important for regular reabsorption, plus
2 enteric coated tablets of calcium EAP
1 ampule containing 400mg calcium EAP—3-4 times per week IV. Inject quickly.

The results of the treatment without the EAP IV are not satisfactory by comparison.

Symptomatic improvement of the illness covers mainly:

* Phosetamin is a German brand name for a product combining the calcium, magnesium and potassium salts of amino ethanol phosphate (AEP)

bladder function
intestinal function
mobility of the upper extremities
facial mobility
generation of body heat
relief from the feeling of complete exhaustion

In contrast, the improvement in the musculature of the thigh is relatively slight in comparison to the above symptomatic improvement.

Spasticity, even with the adductors, is usually considerably lessened, especially by the magnesium EAP (phosetamin). The necessity of prescribing Lioresal is now lessened considerably. About 40% of the ambulators and 60% of the stationeries in MS patients treated receive Ixoten. However, when they are treated with EAP, the Ixoten can be continued—we have used Ixoten therapy continuously for over 1,000 days (50mg daily) without any apparent problems—instead of a limited time, (such as 12-16 weeks) therapy.

On the basis of our clinical observations, the calcium EAP intravenous therapy should be continued for at least four to seven years. We have several patients on our prescription list, that have continued the oral therapy for over 20 years. Frequently, patients will show a remarkable improvement with Ixoten and will ask for a renewal of their prescription. This however, is never the case with Imurek.

At the start of an acute attack, the medicine of choice is triacnolon, or occasionally decadron intrathecal. ACTH should be definitely avoided for the MS patient. It leads to squeezing out of the androgen Fisser-control steroids, and although some improvement may be experienced at first, the effect in the long run is a worsening of the suffering.

The percentage of the MS patients who are objectively and subjectively, in better condition, at the end of two years treatment, is for the American patients, between 90 and 92%. For the European, however, it runs closer to 70%. The strong symptomatic divergence in the USA, for example less optical palliation, and the variation in the therapeutic results, can be best explained by the aluminum theory.

Our conclusion is that from the aspects exhibited, taking into consideration the complete membrane organ systems, from a series of biochemical, laboratory-technical and therapeutic results—all this tied together—indicate that we should consider MS as distinctly an internistic illness, and a neurological illness to a lesser extent.

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