Medical Hypotheses 13: 63-75, 1984

CHRONIC METHANOL POISONING WITH THE CLINICAL AND PATHOLOGIC-ANATOMICAL FEATURES OF MULTIPLE SCLEROSIS

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ABSTRACT

The details of two cases of chronic methanol poisoning are presented. Both patients initially developed clinical symptoms of multiple sclerosis: visual disturbances, intention tremor, reduced abdominal reflexes, impaired coordination and difficulties with walking. After the exposure to methanol had ceased the multiple sclerosis symptoms persisted in patient 1 but disappeared gradually in patient 2 (patient 2 had a history of excessive alcohol consumption, which is a critical fact in this discussion). Ultimately autopsies confirmed this picture: histological examination of patient 1 revealed plaques in the spinal cord, in the stem and in the proximity of the lower horn of one lateral ventricle, whereas no localized demyelination could be found in patient 2. The results are discussed in connection with the theory ("Methanol Hypothesis") that under certain circumstances multiple sclerosis itself is induced by formaldehyde stemming from the metabolism of methanol.

INTRODUCTION

In a recent publication (Henzi, 4), a new concept of multiple sclerosis (MS) was presented (the methanol hypothesis, MeHyp) in which MS is viewed as an essentially toxic--allergic event. Through the intracellular effects of methanol and formaldehyde a repetitive metabolic state causes the formation of minimal amounts of a Schiff base (formaldehyde bonded to $-NH_2$ groups of amino acid side chains) in the CNS. This triggers an immune reaction and antibodies (immunoglobulin-G-antibodies recognized by Lumsden (6) and others) are instrumental in the transform-

ation of the affected tissue into plaques, thus leading to MS. Methanol (from pectins in plant food) and fructose are involved in the metabolic complexities that start the premorbid phase of MS. It is significant that a substantial change in eating habits (increase in fructose consumption) in the early 19th Century preceeded the first appearance of MS in 1818. (Further details, including application of the MeHyp in therapy, may be found in (4).)

In this paper I report on two cases of chronic methanol poisoning that were linked to the appearance of MS and many of its symptoms. It is the first instance in the 160 year history of MS research of anatomic-pathological evidence pointing to the substance lying at the root of the MS problem. Moeschlin (7) affirms that chronic forms of methanol poisoning are recognizable in the two cases. The early histories of the cases were described in detail by Schwarzmann (8) and Dreyfuss (2). The patients could be questioned by the present author and one of them could be examined. Reports of routine autopsy and histological examinations are available for both cases. Here I present the two case histories and the anatomic-pathological and histological reports.

CASE HISTORY 1

(R.G. - born 1899: slightly abridged translation of Schwarzmann (8))

At the time of writing (1933) the technician R.G. was 34 years old and had been superintendent of a laboratory since 1928. During his work R.G. was exposed to vapors of "technical formaldehyde" (12 to 16% methanol) (his exposure was particularly intense and over longer periods compared to other staff members).

Early signs of trouble were intense headache, smarting of the eyes, blurred vision and impeded nasal respiration. In 1930 pains suddenly occurred in the left knee and there was also a "strange sensation" in the left leg, so that the physician consulted suspected sciatica. The pains disappeared after treatment, but a sensation of coldness in the left knee and of heat and pins and needles in the left thigh remained.

After a longish pause the tests involving the technical formaldehyde were recommenced at the laboratory in June, 1931. In September, 1931, he suffered from gastro-enteric disturbances: lack of appetite, and diarrhea alternating with constipation. By December, 1931, he was highly irritable and had bouts of perspiration, respiration trouble, palpitations of the heart, vertigo, blurred and reduced

vision and diplopia. The physician consulted observed pronounced horizontal nystagmus, mild intention tremor, reduced abdominal reflexes, and a wide, unsteady, slightly spastic gait. As these symptoms suggest, the tentative diagnosis was "possible MS".

During December, 1931, the symptoms increased to the extent that R.G. could not get about without being led, vomited when changing position, had splitting headaches, very disturbed sleep, humming in both ears, then sudden loss of hearing in the left ear. His condition became so bad that he had to be hospitalized in January, 1932. On the assumption that his condition was due to a form of poisoning contraced at work, but without knowing the real cause of his illness, R.G. had put in a claim to the "SUVA" (Swiss counterpart to the Workmen's Compensation Board), which requested a first medical report. In essence the report lists: definite nystagmus, but no loss of the fields of vision; ataxia and adiadochokinesia of the left arm; Romberg positive; hyperaesthesia of the left thigh and the soles of both feet, and paraesthesia in the left leg; patellar and achilles tendon reflexes temporarily reduced; cerebrospinal fluid normal. The differential diagnosis considers MS, acute disseminated encephalomyelitis, cerebrospinal syphilis, and methanol intoxication. The diagnosis 'MS' was considered to be the most probable. In consideration of this report R.G.'s claim was rejected.

After R.G. was again exposed to the vapors for a total of 11 hours between July and September, 1932, his health became considerably worse. Schwarzmann (8) reports the following symptoms: vertigo, vomiting, numbness of the whole right half of the body. In addition, palsy of the facial and abducens nerve (left side) and reduction of visual acuity were noted at the beginning of October, 1932. The patient brought a suit against the SUVA in the insurance court and was subsequently examined by Prof. Staehelin (Medical Clinic of the University, Basel). The following is an extract from his report (1933):

> Pendular nystagmus when looking to the right, slight reduction of convergence. Visual acuity normal, apart from a slight refraction anomaly, but a bilateral central colour scotoma could be detected. Reflexes normal, except that the lower left abdominal reflex was absent. Perception for pointed and blunt objects and hot and cold was considerably reduced in a longish zone on the right shank. Ataxia and paresis could no longer be noticed.

As a result of the action the case was accepted by the SUVA as chronic methanol poisoning.

In March, 1946, an ophthalmological control examination was made by Dr. Buerki (Basel). The relevant part of his report is:

> Horizontal pendular nystagmus, particularly when looking towards the left. Partial damage to retina and optic nerve with reduction in visual acuity (0.7 bilaterally corrected), central or paracentral color scotoma, acquired disturbance of color vision, a lowering of dark adaption and possibly an acquired alteration in distance perception in bilateral vision. Possible temporal pallor of the papillae - can not be judged properly because of marked physiological excavation.

From a neurological report by Prof. Georgi, 1953:

> ... Sense of smell less acute left than right. Paresis of the right abducens nerve, horizontal pendular nystagmus, slight palsy of left facial nerve. Impaired hearing in right ear. Upper limbs: possible slight weakening in left arm; left thumb can be only partially opposed to the little finger. Abdominal reflexes lacking. Lower limbs: bilateral spasm, position test impossible on both sides; advanced paraparesis of both legs: motility, gross strength and perception of vibration very considerably reduced.

Dr. Schnyder, who treated R.G. the whole time, told me that the neurological findings showed a gradual deterioration. Most outstanding were fatigue (adynamia), insomnia and spasms in the lower limbs.

I visited R.G. in 1967. He declined to be examined by me, but readily answered questions. R.G. lived practically as a teetotaler, he had fruit from his orchard and ate a lot of sugar. As mentioned in detail in Henzi (4), a close correlation has been found between the ingestion of fructose together with methanologenic pectin (as is found especially in incompletely ripened fruits) and the incidence of MS*.

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* Human metabolism is well adapted to harmlessly degrade methanol derived from plant foodstuffs and its toxic metabolites. Catalase, alcohol dehydrogenase and the C_1-pool (tetrahydrofolic acid) are involved. The lifetime of at least one toxic metabolite, formaldehyde, may be prolonged under certain conditions, however: glyceraldehyde competes with formaldehyde for degradation by alcohol dehydrogenase. One source of such glyceraldehyde is ingested fructose. The introduction of sugar production from sugar beets around 1804 soon resulted in high levels of fructose consumption. The fact that MS was not found until after this development (first known report in 1818) has been overlooked in MS

Under these circumstances a fructose loading may at times be reached such that methanol and its metabolites are no longer effectively cleared from the body. According to the methanol hypothesis, there is then a risk of being sensitized to formaldehyde-altered proteins, thus leading to MS. Patient R.G. belonged to this category. Furthermore, in addition to the direct effects of the technical formaldehyde (irritation of mucous membranes, etc. by formaldehyde), the repetitive inhalation of the methanol component would further increase the blood concentration of methanol and thus enhance the likelihood of initiating MS (see Henzi (4) for more details).

I have extracted the particulars of the history of his invalidism from the final report of the "Kreisspital (District Hospital) fuer das Freiamt" (Chief medical officer: Dr. Schmid):

> Up to 1943 relatively free of handicaps. 1945 - dragging feet. Until 1945 the patient could cycle, later he used a car. In 1955 he could walk with the help of a cane. By 1958 he could get around with difficulty on 2 canes. Later he could no longer get around, but was still able to sit. Bedridden from early 1973. Death from aspiratory pneumonia in 1974.

Pathological Anatomy

The autopsy report (Cantonal Hospital, Lucerne - Prof. Aufdermaur: Feb. 27, 1974), contains the following:

> Patient R.G. - Anatomic-pathological diagnosis and findings: Lacunar condition of the brain, pneumonia, nephritis. "... The brain shows a principally perivascular degradation of the parenchyma. This is very marked in the ganglia of the brain stem. I can not ascertain any findings in the brain or spinal cord which would favor a diagnosis of multiple sclerosis."

In order to answer the question as to whether the changes in the CNS are due to an intoxication through formaldehyde or methanol vapors, Prof. Aufdermaur proposed that

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research. A further increase in fructose consumption has been found in all MS countries since the 1970's. The history of MS parallels and reflects these developments. Furthermore, as nutritional habits change, MS appears among hitherto apparently immune populations (e.g., Japanese immigrants in Hawaii or Vietnamese in France). Many contemporary reports show the deleterious aspects of high fructose consumption (9, 10). There is good evidence for the disturbance of methanol degradation by fructose (4).

the opinion of a neuropathologist be obtained. From the report by Prof. J. Ulrich of the Pathological Institute of the University Hospital, Basel (abridged translation):

Macroscopic: no gross dilation of the ventricles. No conspicuous changes. The cerebral cortex is 2-3 mm thick everywhere. Near the left lower horn of the lateral ventricle there is a possible MS plaque of approximately 4 mm diameter. Spinal cord unremarkable.

Microscopic: lateral pyramidal tract (lumbar) obviously depleted on both sides. In a frozen section of the thoracic region of the spinal cord the frontal part of the posterior column is clearly pathological with sudan staining: very clearly demarcated plaque which is delimited in front by the posterior horns and the grey commissure. No myelin sheath can be detected within this region. There are also no fat-granule cells and no infiltrates.

Medulla oblongata: proliferation of the astroglia on one side in the lateral portions of the medial lemniscus, transgressing into the intramedullar portion of the hypoglossal nerve as far as the reticular substance. Staining for myelin sheath appears less intense in the area of glial proliferation. This zone may be a so--called shadow plaque.

Pons at the level of the pontine arms: a lentil-sized plaque is visible in the upper part. Adjacent to the main plaque is a smaller plaque, which is likewise sharply delimited, with a central vein and a few individual ganglion cells in the interior.

Cerebellum: no sign of pathological change, with the exception of a single lamella.

Pons further rostral: the section passes through the locus ceruleus. On the one side it is in the middle of a completely demyelinated zone. This plaque is also fully developed, i.e., no definite myelin sheath can be demonstrated to occur in the interior. Of interest is a ventral zone attached to the plaque, in which the myelin sheaths are sparser but not completely missing.

Brain: lentil-sized, completely demyelinated zone near the lower horn. Further somewhat narrower demyelinated zones, also directly subependymal. Adjacent to the first-mentioned lentil-sized zone is an incompletely demyelinated area. Right occipital lobe: incompletely demyelinated strips, partly sharply, partly only diffusely defined. Cross-section through the thalamus:

there is only one definite plaque near the lower horn. Frontal lobe: a possible demyelinated zone in the white matter.

DIAGNOSIS: Chronic multiple sclerosis which has run its course, with plaques in the spinal cord, in the stem and in the proximity of the lower horn of one lateral ventricle.

Discussion of Case 1

1) Causal connection. The sequence of exposure to formaldehyde/methanol vapors and the observation of pathological symptoms is compatible with the MeHyp. R.G., who has been described as intelligent and devoted to his work, was healthy previous to the poisoning episode. Some of the symptoms can be ascribed to the known toxic effects of formaldehyde, such as smarting of the eyes, and impedence of nasal respiration. Surface effects of formaldehyde on mucous membranes can explain this, and perhaps the intense headaches could be explained in this way as well. The gastro-intestinal disturbance noticed in September, 1931, i.e., lack of appetite, alternating diarrhea and constipation, appear to have a toxic cause.

2) There is an obvious sequential relationship of symptoms of a toxic nature in a first phase, followed by a second phase in which the symptoms of multiple sclerosis, i.e., of the allergic reaction (commonly called "autoimmune reaction"), become ever more visible.

3) Quantitative considerations. The patient inhaled only vapor. Even if we assume that the air in the working atmosphere was saturated to the extent that smarting of the eyes could still be stood, the quantities inhaled - the relevant factor in considering the aetiology of R.G.'s chronic illness - were quite modest.

Humperdinck (5) remarked that while the poisonous but sublethal oral dose is considered to be 5-10 g of methanol, there is no definite information as to the quantity of methanol vapor which would have to be inhaled to cause acute poisoning. He noted that the inhalation of highly concentrated vapor, such as would be necessary to absorb the amount corresponding to the oral dose, is hardly possible, as a concentration approaching 5 vol% (65 mg/l air) causes intense irritation.

CASE HISTORY 2

(St.E. - born 1885: translation of Moeschlin (7): Dreyfuss case)

During 1940 (at 55 years of age) and 1941 St.E. worked in a garage and had regular contact with methanol, as he had to fill this substitute fuel into tanks. Several times a day he had to suck at a hose dipped into methanol to start the siphoning process, and some methanol always got into his mouth. Simultaneously he was also exposed to methanol vapors.

After 6 months at this work he felt the first symptoms: pains in the thorax and an intense headache in the back of the head. He noticed that his voice had become weaker, and gradually additional symptoms, such as excruciating and burning pains in arms and legs, and paraesthesia, developed so that he had to seek medical advice. He was never paralyzed, but had to walk with the aid of a cane.

He experienced symptoms on and off until he was admitted to the Cantonal Hospital (Zuerich) on October 31, 1941. He complained about burning pain in the fingers and toes and of a sensation as if the back of the head were swollen. He also mentioned the decreased strength of his voice.

In the hospital an examination was made for a report to the SUVA, which in due course accepted the claim as a case of occupational methanol poisoning. The neurological status at that time was:

- -eyes - no nystagmus; left papilla somewhat paler than right, but not necessarily pathological; slight enlargement of the blind spot in the right eye; relative scotoma.
- -trigeminal nerve exit sensitive to pressure.
- -sense of taste and smell missing on the left side; bilateral acoustic neuritis; paresis of the thyreoarytenoid muscles (more pronounced left than right) with no history of hoarseness; speech low-keyed, halting.
- -arms - strong reflex, diadochokinesia poor, especially left; intention tremor in finger-nose test; ulnar nerve pressure-sensitive (as was the sciatic nerve, but the possibility of peripheral causes was not investigated further); strength good, but reduced gripping power.
- -lower limbs - knee jerk exaggerated (moreso right than left); ankle jerk brisk; no Babinski reflex; Romberg's test: tendency to fall backwards to the

right, but can be controlled.

-micturition disturbed from 1940 to summer, 1945; incontinent.

-abdominal reflexes feeble and exhaustible; reduced feeling on the left side, hypoaesthesia, hypoalgesia, thermo-anaesthesia; gait slow, lack of impulse; little coordinated swing of left arm; increased salivation; tremor when resting, as with Parkinson's syndrom.

Supplementary information translated from Dreyfuss (2): St.E. had been employed in a brewery a few years before the methanol episode. At that time his daily intake of beer had been 4-5 liters. He consumed very little sugared food. A clinical follow-up examination could be made in 1968. His neurological status was apparently normal, pathological symptoms were no longer observable even on ophthalmological examination in the Eye Clinic (Basel). St.E. stated that he had consumed about 1200 capsules of vitamin B-complex and that he used to rest a lot even after he had ceased to have contact with methanol. From 1948 onwards he was again able to walk normally (i.e., without a cane). Died in 1974.

Autopsy of the CNS

Pathological Institute of the University Clinic, Basel, (Prof. J. Ulrich):

"Advanced senile atrophy of the brain. Extensive obliteration of the optical nerve with fluid-filled cysts bilaterally. According to the clinical reports the atrophy of the optical nerve may be a residuum from a methanol intoxication 32 years previously."

Histological examination by Prof. J. Ulrich:
In spite of normal brain weight (1260 g) there was a distinct atrophy of the brain with gaping furrows and almost crestlike convolutions. All lobes were uniformly affected by the atrophication. There was a corresponding internal hydrocephalus. No plaques could be observed anywhere. The section of the spinal cord showed small cysts on many spinal ganglia.

Painstaking microscopic examination showed atrophy of the cortex with many senile bodies and isolated instances of Alzheimerian alterations in neurofibrils. The white matter was atrophied but no demyelination was seen. Only the optic nerve showed somewhat thinner myelin sheaths. There was definitely no localized patch of demyelination, merely an atrophy.

DIAGNOSIS: Senile atrophy of the brain. The change in the optic nerve could also be interpreted as an age--induced atrophy, but it can not be excluded that there may be a discrete residue of the methanol poisoning episode. It is absolutely certain that this patient was not suffering from multiple sclerosis.

Discussion of Case 2

Why was no sign of MS found in the autopsy of St.E. in spite of the fact that his exposure showed a similar pattern to the case of R.G.? The toxicologist JP von Wartburg (personal communication, 1974) explains the different reaction to methanol as follows:

"... With this considerable ethanol consumption we must presume that he had the changes in the liver normal in alcoholics. Amongst other changes this means that the microsomal ethanol oxidizing system (MEOS) had been induced (increased enzyme activity). In this system catalase and other enzymes oxidize alcohol and, in contrast to alcohol dehydrogenase, these are inducible enzymes. MEOS oxidizes methanol as well as ethanol. Thus it is likely that St.E. oxidized methanol rather faster than individuals in whom the MEOS had not been induced."

This allows the conclusion that in St.E. methanol as such had its effect (narcotic effect in cells), due to its distribution in the CNS as typical for aliphatic alcohols. This caused clinical symptoms of a MS which actually figured in the differential diagnosis. Because of St.E.'s induced MEOS the delay in the degradation of methanol in the formaldehyde stage did not occur. Thus the change in conformation of membrane proteins - formaldehyde bonding to the $-NH_2$ groups of the side chains of the amino acids - did not take place and so the immune reaction was not induced. There were therefore apparent MS signs and symptoms, but no plaques developed that would be observable in anatomic-pathological examination.

OTHER CASES OF CHRONIC METHANOL POISONING

A few other cases have been described in the toxicological literature (see Humperdinck (5), Burk (1) and Hansohm (3)). A routine autopsy was only made in the last mentioned case, but no histological examination was performed. The patient died after many years of chronic exposure, wnich had led to atrophy of the optic nerve. (Chemical analysis indicated he committed suicide by a purposely inflicted acute methanol poisoning.)

That findings such as in case 1 (R.G.) are rare may have several reasons:

i) Methanol is classed as a poison and the legal requirements are such that it is infrequently used. Present use is in laboratories and as fuel in model aircraft motors. It is also being considered as an additive for gasoline for motor cars.

ii) The disturbing factor of excessive fructose consumption, which delays the degradation of methanol ingested in food, is not always present (discussed in MeHyp, ref. 4).

iii) The "antidote" effect of frequent ethanol consumption. Ethanol basically is not an antidote. It prevents the degradation of methanol to formaldehyde and thus provides protection from the toxic effects of methanol metabolites in acute methanol poisoning. The same appears to hold for the sensitization to MS caused by formaldehyde.

iv) Cases do occur, but an autopsy is either not performed or performed without histological examination. (See case 1 (R.G.) where the plaques could be seen only on histological examination.)

The effects of acute methanol poisoning are also of interest in the present discussion. Although Orthner (11) suggested more than thirty years ago that the survivors of such incidents be examined, only the report on two cases by McLean et. al. (12) is known to the author. Surprisingly they show necroses in about the same regions as in case R.G. (this paper), with the exception of the findings on the optic nerves, which were almost devoid of myelin. Judging from other reports, this presumably resulted from a secondary wallerian degeneration following damage to the retinal ganglion cells.

Large doses of methanol yield necroses while small doses may result in demyelination under certain conditions. Putaminal necroses are apparently found only with large doses. A detailed discussion of these questions is beyond the scope of this article, but further references are to be found in McLean et. al. (12).

DISCUSSION

The observations in the two cases presented permit the following alternate views:

i) fortuitous, simultaneous occurrence of chronic methanol poisoning and MS in these two individuals

ii) chronic exposure to methanol (e.g., repetitive occurrence of minute quantities of methanol in the individual's circulation) leads, when certain other factors are simultaneously present, to MS (i.e., methanol is a common cause of both afflictions).

Coexistent diseases as implied by the first possibility are thought to be unlikely. To obtain a numerical estimate of the probability of such an occurrence we may assume that the 5000 observed cases of MS in Switzerland have occurred over a 30 year period at a constant incidence and that in the same period 15 cases of chronic methanol poisoning occurred. For the 6,000,000 inhabitants of Switzerland the probability of acquiring MS is thus 5000/6,000,000 = 1/1200 in 30 years. The probability of methanol poisoning is 15/6,000,000 = 1/400,000 for the same period. Free combination of the probabilities gives 1/1200 x 1/400,000 = 1/480,000,000. That the observed frequency (1/6,000,000 or 2/6,000,000, depending on whether or not the case of St.E. is included) is eighty (or 160) times higher than this, favors the view of a causal connection. (The assumption of 15 cases of chronic methanol poisoning may be questioned, but it is certainly an extremely infrequent event and this number would seem to be a reasonable estimate.)

The methanol hypothesis of MS postulates that formaldehyde stemming from the metabolism of methanol, if not degraded, modifies proteins of the myelin sheath. Due to the low rate of turnover of such protein there may, under suitable conditions, be sufficient time for the immune system to recognize this altered protein. This leads ultimately to sensitization and an attack by IgG. Demyelination and the formation of plaques results. It should be noted that exposure to exogenous formaldehyde is not expected to induce MS. Because of its more hydrophilic character, formaldehyde can not easily cross the membrane barriers to reach the ultimate target. Most other methylating agents are likewise unlikely to be active at the sites of the observed plaques. Methanol, in contrast, can permeate the blood-brain-barrier and then be degraded to yield formaldehyde in situ.

CONCLUSIONS

The case histories discussed in this paper show that chronic methanol poisoning can cause the symptoms of multiple sclerosis and even, under suitable circumstances, induce the disease itself. These findings support the "methanol hypothesis" of multiple sclerosis, which views MS as a toxic-allergic event resulting from the action of formaldehyde stemming from ingested methanol on the myelin sheath.

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