EXPERIMENTAL MEDICINE. – *The psittacosis virus in the etiology of multiple sclerosis.*

Report (*) by Mr. Paul Le Gac, Mr. Frank Wolffaert, and Mr. Emile Arquie, presented by Mr. Jacques Trefouel.

*Abstract:* The psittacosis virus is one of the etiological factors in multiple sclerosis. This condition is not the result of a specific infectious agent, but instead primarily the result of a group of angiotropic infectious agents (the *Rickettsieae*), which, because of their endothelial affinities and their toxins, cause (over the course of a series of latency and resurgence phases) cell anoxia, which is the pathogenic process that lies at the base of multiple sclerosis.

On several occasions we have emphasized the importance of the role played, in the pathogenesis of multiple sclerosis, by bacteria belonging to the order *Rickettsieae* [1].

We initially found that this role is played primarily by one of the superfamilies in this order, i.e., the *Rickettsieae* (rickettsia). We subsequently found that this role also extends to another of the superfamilies in this order, i.e., the *Chlamydiaceae*, and, in particular, to the genus *Rakeia* (which is the ovine abortion virus) [2].

Today we are adding to this pathogenic group a new agent that is also a member of the order *Chlamydiaceae*, but that belongs to the genus *Bedsonia*, i.e. the psittacosis virus.

The epidemiology guided us toward this pathogenic agent during the course of investigations that were undertaken in connection with our research on the etiology of multiple sclerosis.
The micro–agglutination reactions to the rickettsias that took place with the serum of two patients with multiple sclerosis remained obstinately negative, even after reactivation. Thus, we proceeded with an epidemiological investigation of the activities of these two subjects.

Through this investigation, we learned that each of these subjects owned a breeding group of parakeets imported from Brazil, and that on several occasions these breeding groups had been affected by a disease of an indeterminate nature. This very important finding led us to the psittacosis.

The serum of each of these two subjects was examined by Prof. Jean Jadin, at the Institute of Tropical Medicine of Antwerp, for the T–13 psittacosis strain (which had been isolated by Prof. Brenno Babudieri, of Rome, from a parrot imported from Argentina). The results of this examination were strongly positive for this antigen.

The observations of these two patients are summarized below.

1. Mr. Maurice Q., a Belgian citizen, 46 years of age. Multiple sclerosis was manifested in 1955 by transient retrobulbar neuritis. In 1956 he became bedridden. As of November 1961, [he had been] totally quadriplegic for three years.

   The serodiagnosis was strongly positive for the T–13 strain. Antibiotic treatment and alginated baths were followed, within a few months, by a spectacular improvement.

   In May 1962, Mr. Q. was walking normally. He was able to discard all assistive devices, and soon afterward went back to work as a freight–truck driver.

   Epidemiologically speaking, the presence of a breeding group of parakeets in his home provided a clear explanation of the contamination.

2. Mr. Albert Van S., a Belgian citizen, 31 years of age. Multiple sclerosis appeared for the first time in 1951, in the form of left retrobulbar neuritis, which disappeared within two months.
In 1956, mild weakness of the right leg appeared, with a tendency toward tremulousness. The disease progressed slowly but implacably, with a spastic syndrome affecting the lower limbs, the arms, and the left hand, in which all sensitivity was lost. Walking became painful and then impossible without assistance.

In May 1961 the serodiagnosis was clearly positive for the T–13 strain.

Antibiotic treatment, in association with thalassotherapy, produced a transformation of the patient’s general state of health, with the disappearance of all pathological symptoms. Within a year the patient was able to return to his job, which requires four daily bicycle trips that the patient was able to complete without becoming fatigued.

Like the patient described above, this subject also had an aviary of parakeets from Brazil.

Based on these observations, which were subsequently confirmed by others, it was clear that the psittacosis virus can be considered to be one of the pathogenic agents of multiple sclerosis.

In point of fact, therefore, multiple sclerosis is not caused by a single specific infectious agent, but rather by any one of a group of strongly angiotropic agents which, thanks to their endothelial affinities and their toxins, produce (over the course of a series of latency and resurgence phases) the cell anoxia that constitutes the fundamental pathogenic process of this condition.

Through its two superfamilies *Rickettsiaceae* (i.e., the rickettsias) and the *Chlamydiaceae* (i.e., the para–rickettsias) the order *Rickettsiaeae* accounts for the principal agents, among which the members of the so–called “TPL group” (trachoma, psittacosis, and lymphogranulomatosis) hold a predominant place.

All of these bacteria also meet the conditions required by J.B. Sabin of Cincinnati, who posited, in principle, that the pathogenic agent for multiple sclerosis should, above all:
a. Be capable of residing (in the latent state) in the central nervous system, and of experiencing periods of activity provoked by physiological or non-physiological disturbances; and

b. Be capable of attacking the glial cells that regulate the formation of myelin.

However, the enterobacteria must also be linked to this principal etiology of multiple sclerosis, because of their endotoxins, which are highly angiotropic and thus capable of causing vascular lesions that in turn lead to cell anoxia.

Regardless of the infections agent, the effect of the toxin is dominant. It plays a predominant role in the genesis of remissions. When the pathogenic agent displays a limited degree of vitality in the organism (as is the case with the enterobacteria), its toxins are exhausted. If no final or determining lesions are already present, then the remission becomes definitive and healing takes place.

However, when the pathogenic agent is present in the organism for an extremely long time, as demonstrated by Edmond Sergent in connection with rickettsias, quite different events take place. Under these circumstances, a series of relapses and remissions ensues, which is often dictated by the seasonal cycle of these factors. No final remission can be achieved as long as these viruses retain their virulence.

The latency that is so characteristic of these pathogenic agents, alternating with phases of resurgence, is the reason why multiple sclerosis has remained an enigma for such a long time, from both the etiological and pathogenic points of view.

Thus, the infectious theory of multiple sclerosis posited by Pierre Marie [3] has been confirmed, in a manner that is moreover quite encouraging, because it allows us to envision a therapeutic approach that is both active and curative, and that has also already yielded excellent results.
In point of fact, we know that broad–spectrum antibiotics (including terramycin, aureomycin, ledermycin, and typhomycin) represent a weapon of choice against rickettsial agents, and have been shown to be particularly effective against their acute, as well as chronic, manifestations.

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(376, avenue du Marechal–Lyautey, Saint–Raphael, Var)