A MOST MYSTERIOUS DISEASE:
A CULTURAL HISTORY OF MULTIPLE SCLEROSIS, 1868-1958

by

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I
THE HISTORIOGRAPHY OF DISEASE AND THE HISTORICAL PROBLEM

Charles Rosenberg argues that, "disease is both a fundamental substantive problem and analytical tool in the history of medicine and the social sciences." Disease is a "multidimensional sampling device for the scholar concerned with the relationship between social thought and social structure."[1] The symptoms of a disease are translated by cultural interpretation into a disease nosology. The disease concept then becomes an actor in the process of negotiation between: 1) physicians and patients; 2) patients, families and society; and 3) medical institutions, medical ideas, and medical practices. This places Rosenberg within the latest methodological thinking within American social history. Alice Kessler-Harris writes that, "the best social history attempts to integrate new research in institutional structures with consciousness and ideology in a way that creates understanding of broader political process and of the tensions that ultimately yield change."[2]

The period from the late 1960s through the 1980s was the height of the social constructionist period in the writing of the history of disease. Charles Rosenberg argues that the primary foci in the study of the history of disease since the late 1960s have been: the deconstruction of disease; the use of disease definitions as a tool of social control; the social experience of biological events like pregnancy, childbirth, and epidemics; the connections between public policy, the demographic revolution in the early twentieth century, and broad demographic patterns of morbidity and mortality (e.g. the Mckeown

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[1] Charles E. Rosenberg, "Illness, Society, and History," in *Framing Disease Studies in Cultural History*, ed. Charles E. Rosenberg and Janet Golden (New Brunswick: Rutgers University Press, 1992), xxiii.

[2] Alice Kessler-Harris, "Social History," in *The New American History*, ed. Eric Foner (Philadelphia: Temple University Press, 1990), 180.

thesis); and the new ecological history that emphasizes the impact of disease on history.[3]

Rosenberg argues that the social constructionist school has tended to underestimate the importance of the biological event of disease as an actor in the process of the cultural attempt to understand that event within a specific set of social structures. The new history of disease has sought to integrate and problematize the earlier histories of disease as celebrations of conquest. Ironically, most of the diseases that historians have analyzed such as, tuberculosis, yellow fever, smallpox, and cholera, have tended to be ones in which the biological event of the disease is relatively straightforward and diseases where a cure was found. In contrast to these previous studies, this work will examine the history of a disease where the biological event and the pathological symptoms attendant to it are relatively unstable. In this way we can query Rosenberg's idea that the particular biology of a disease can become an actor in the process of cultural negotiation. Multiple sclerosis is an excellent sampling device for this because the biology of the disease itself shifts, mutates, and unpredictably remits.[4] [See Table XI.] The polymorphic nature of MS informs the next question: how can a disease can suddenly erupt into public consciousness when there is no dramatic epidemic or identifiable epidemiological change.

To make the historical problem clearer, consider this: in 1922 the total number of

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[3] Charles E. Rosenberg, "Introduction: Framing Disease: Illness, Society, and History," in *Framing Disease: Studies in Cultural History*, ed. Charles Rosenberg and Janet Golden (New Brunswick: Rutgers University Press, 1992), xv-xvi.

[4] I do not mean to suggest that humans have access to an unmediated essential biology or that if we just remove the cultural and social layers we can then see the disease as it is. What I do suggest is that there are limits to the cultural appropriation of our bodies and the natural world. Beyond the limits of this appropriation what I call biology might be understood as the unappropriated complex chaos of the natural world which, like the return of the repressed, can impact our linguistic mediations by suddenly inserting unsettling mystery into these cultural constructions precisely because we do not know.

MS cases in the United States was estimated at between 511 and 10,057 with a rate of 10 per 100,000. This sense that MS was a rare disease remained as late as the mid-1940s. Suddenly, in the 1950s there were estimates of between 250,000 to 300,000 cases with a rate of 166 per 100,000 in the United States and physicians and lay persons alike considered it a pressing medical problem. [See table I.] It seemed that multiple sclerosis had come out of the blue in terms of public consciousness and in terms of being a priority concern of the medical community. As I will show, it was probably not that there were suddenly more multiple sclerotics in terms of people suffering from the underlying condition of the destruction of the myelin sheath and scarring of the central nervous system. If there were suddenly more MS cases it was one of the greatest unseen epidemics of all time. It was not that there was any new understanding of the etiology of the disease or its cure nor were there any new breakthroughs with regard to multiple sclerosis in the 1950s. The historical problem around which this essay revolves concerns explaining how the perception of the scope of the problem changed so rapidly from the mid 1940s to the mid 1950s. The answer is a complex one and begins in an analysis of the decades before the 1940s to understand how this happened.

II
WHAT IS IT?

In a May 1954 article in the *Saturday Evening Post,* a thirty-one-year-old multiple sclerosis patient Robert Grant, Jr. told the story entitled, "I've Got the Most Mysterious Disease." Grant was a thirty-one-year old veteran of World War II who was admitted to the VA hospital in Boston in 1948. Grant framed the question that had challenged neurologists for over eighty years.

> Just what is multiple sclerosis? It is a tragic fact that I can explain its pathology to you almost as competently as any doctor in the field. This is an indication, not of my erudition, but of how little is known concerning

> this disease . . . By mail I established contact with the chief neurologists of several clinics. Sometimes they thought my questions naïve, but more often they expressed surprise at my intimacy with such terms as meningeal infiltration and perivenous lesions.[5]

Today, as in 1954, there is no cure and no clear understanding of the disease's ultimate cause. To begin this section it is enough to illustrate the basic disease process and the possible clinical presentations so that one can understand how the peculiar biology of this disease problematized clinical and laboratory diagnosis before the age of the MRI.

As it is understood in 1995, multiple sclerosis is an auto-immune disease in which T lymphocytes, B lymphocytes, and macrophages attack the myelin sheath that surrounds the axon. Macrophages directly attack the myelin by presenting fragments of the protein on their surface for T cells to recognize. The B lymphocytes produce antibodies that destroy the oliogodendrogial cells, which normally repair myelin. The T lymphocytes mistake an antigen produced by the body for a foreign antigen and attack the body tissues that contain the self-antigens. The destruction of the myelin sheath in the brain and the scarring that ensues inhibits the transmission of nerve impulses down the axon.[6] The symptoms exhibited by patients vary widely depending on where the sclerotic patches are and how often the patient has been attacked. Patients may experience tingling sensations, pain in the extremities, tightening sensations around the waist, vertigo, loss of muscular control, partial paralysis, temporary blindness, diplopia, scanning speech, nystagmus and emotional changes. [See Table II.] Victims may experience all, some, or none of these symptoms in various combinations which may remit for long or short periods or may slowly worsen. Because of these widely variable pathological symptoms, clinical

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[5] Robert Grant, Jr., "I've Got the Most Mysterious Disease," *Saturday Evening Post* 226 (22 May 1954): 120-128.

[6] Lawrence Steinman, "Autoimmune Disease," *Scientific American* (Sept 1993): 108-109.

diagnosis of multiple sclerosis has long been difficult. The peculiar biology of this disease meant that certain social structural forces and scientific advances had to come together in order for their even to be the possibility of a transitory glimpse at the construct, multiple sclerosis. Specifically, tensions emerging between laboratory medicine and clinical medicine could inhibit or enhance the possibilities of seeing this condition.

The discovery of the modern pathological, histological, and nosological descriptions of multiple sclerosis, or *sclérose en plaques*, is usually credited to the famed French neurologist Jean Martin Charcot with his "*Histologie de la sclérose en plaques* (1868)."[7] Charcot began his lecture, of which the seminal article is the record, by admitting that "up until now we only knew the lesions of *sclérose en plaques* by their exterior side that is visible to the naked eye." Charcot then went one to describe the histologic technique used to uncover the microscopic pathologic anatomy of the sclerotic patches.

> It will be, I believe, advantageous to inaugurate this study with an examination of the narrow, transparent slices, transversely cut in the spinal sections that have been conveniently hardened in a solution of chromic acid, and colored by carmine. For this purpose Carmine is a valuable reagent. Thanks to it, certain elements which have the property of coloring themselves under its influence, with a tint more or less lively, are put in relief, while the others retain their ordinary aspect. Thus the ganglion cells, their cores, their nuclei and also the prolongations of these cells, strongly color themselves under the influence of this reagent. The conjunctive neuroglia advantageously colors itself equally throughout its length in a less pronounced manner. And, by this treatment of the nerve ducts, only the axil cylinder takes the color of carmine, and the myelin envelope resists completely its action.

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[7] Jean Martin Charcot, "Histologie de la sclérose en plaques," *La Lancette Française Gazette Des Hopitaux Civils et Militaires* 41, no. 140 (1 Dec 1868): 554. This seminal article appeared in three installments: part one as cited above, part two in *Gazette Des Hopitaux* 41, no. 141 (3 Dec 1868): 557-558 and part three in volume 41, no. 143 (8 Dec 1868): 566.

Through this technique Charcot was able to describe the destruction of the myelin sheath, the relative sparing of the axons, the overgrowth of glial tissue, and the accumulation of fatty globules around the blood vessels at the edge of a sclerotic patch.[8] Charcot wrote, concerning the histological alterations of the spinal cord with disseminated scleroses, that:

> the description of these alterations which we are going to present to you will be based above all on the investigations to which M. Vulpian and myself have devoted ourselves for a long time. We will have occasion to profit from, after verifying, the researches made earlier, or from that time, on the same subject, by Valentiner, Rindfleisch, Zenker and above all by Frommann, who with respect to the examination of a small fragment of the spinal cord, wrote a large book accompanied with remarkable plates and rich in valuable documents.[9]

Charcot is referring to work done in Germany from 1856 to 1868 with the bulk of it done in the 1860s. In Charcot's mind his work was primary research not dependent on the studies being conducted by the Germans though they might be considered parallel. French and German neurologists were exploiting the new tissue staining techniques developed in the service of the dye industry in Germany and pioneered by Virchow. The emergence of Charcot's work on multiple sclerosis specifically in the 1860s was partially rooted in Charcot's competition with the German research community over the fruits of the new staining techniques. However, Charcot argued that Frerich of Breslau (1849), who described what were thought to be several cases of spinal sclerosis, mixed in a number of cases that were not *sclérose en plaques*. Charcot maintained that Valentiner,

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[8] Charcot, *Histologie,* parts two and three. McAlpine, *Multiple Sclerosis*, 2.

[9] *"La description de ces alterations que nous allons vous présenter sera fondée surtout sur les resultats des investigations auxquelles nous nous sommes livrés depuis longtemps M. Vulpian et moi. Nous aurons en outre plusieurs fois l'occasion de mettre à profit, après contrôle, les recherches faites antérieurement, ou depuis lors, sur le même sujet, part Valentiner, Rindfleisch, Zenker et surtout par Frommann qui, à propos de l'examen d'un petit fragment de moelle, a écrit un gros livre accompagné de planches remarquables et riche en documents précieux."* Charcot, *Gazettle Des Hopitaux*, 557.

Rindfleisch, Zenker, and Fromman never clearly coordinated the clinical manifestations of multiple sclerosis with the sclerotic patches. And in the few instances when a loose correlation was made, the patches were thought to result from tertiary syphilis or a generalized myelitis. The Germans did not construct a new nosological category. Charcot did not consider his work dependent on theirs and further that the German work was not quite right because they did not coordinate clinical symptoms with the pathological anatomical laboratory. Many subsequent neuroscientists, such as Walter Timme (1922), McAlpine (1955), and W.I. McDonald (1993)received a mythologized linear research tradition that sees Charcot's research as being dependent on previous German work. It is probably more accurate to say that the two communities were proceeding independently and contemporaneously with the new staining techniques in the 1860s and saw themselves in competition for scientific success. Franco-Prussian completion in the years around 1870 hardly seems surprising. It was competition ***between*** these communities in the 1860s to exploit the new tissue staining techniques that is part of the reason that *sclérose en plaques* emerged specifically in the 1860s as a concept for scientific debate. The reason that Charcot and the French school were able to initiate the beginnings of what we consider the modern understanding of multiple sclerosis, rather than the Germans, had to do with the differences between the two groups with respect to the relationship between the clinic and the pathological laboratory.

German research in this area was hampered because it was decentralized in many different locations and the researchers concerned with this question frequently moved from one institution to another. [See Table III.] At the *Salpêtrière*, Charcot and Vulpian had access to a relatively stable group of patients who could be studied for an extended period of time and then autopsied. Because of the fluid, competitive and decentralized nature of the German medical institutions these combinations of long-term clinical observation, large numbers of patients, and autopsies were harder to achieve though not

impossible. For example consider the careers of four of the German researchers considered early contributors to the study of multiple sclerosis. George Theodor Valentiner was awarded his medical degree in 1843 at Kiel with the dissertation "Questiones duae de typho." From 1849 to 1850 he was Provisor (Dispenser) then Head Physician at a navy hospital in Kiel and he was a Privatdocent. Later he was a general practitioner in Pyrmont till his death in 1877.[10] George Eduard Rindfleisch took his medical education in Heidelberg, Halle, and Berlin from 1856 to 1860 under Virchow. He went to Heidenhain after Breslau in 1861 and qualified as a university lecturer in pathological anatomy. In 1862 he was appointed a lecturer in pathological anatomy in Zurich and was promoted to assitant ordinarius professor in 1864. In 1865 he was named ordinarius professor of pathological anatomy in Bonn. [11] Friedrich Albert von Zenker studied in Leipzig from 1843 to 1847, in Heidelberg from 1848 to 1849, in Vienna in 1850, and graduated from Leipzig in 1851 with a specialty in pathological anatomy. From 1849-1851 he was an assistant physician at the Leipzieger Georgen-Hospital. He took charge of the position of *Prosector* at the state hospital in Dresden in 1851 and from 1853 to 1855 was a lecturer, then later a professor of general pathology and pathological anatomy in the Surgical-Medical Academy in Dresden till 1862. He then became ordinarius professor in Erlangen for over thirty years.[12] Frommann took his medical education in Jena, Göttingen, Prague, and Vienna and graduated with honors in 1854. From 1856 to 1858 he was an assistant physician at the medical clinic in Jena. From 1858 to 1860 he was a house physician at the German hospital in London. From

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[10] *Biographisches Lexikon der hervorragenden Ärzte aller Zeiten und Völker*, ed. E. Gurlt and A. Wernich (Berlin: Urban & Schwarzenberg, 1934), 693.

[11] Ibid., 52.

[12] Ibid., 1034-1035.

1861 to 1870 he was a general practitioner in Weimar. From 1870 to 1872 he was a privatdocent in Heidelberg and from 1873 to 1874 a privatdocent in Jena. He was awarded a professorship in Jena in 1875.[13] Though this was a period of rapid growth in the field of pathological anatomy in Germany, the institutional structures meant that certain pathological conditions would be more amenable to detailed study than multiple sclerosis. [See Table IV.] Only in Paris did the institutional social structure allow the pathological laboratory and the clinic to be combined so that Charcot could describe both the pathological lesions and the clinical symptoms. The Germans did excellent work in their pathological laboratories but the researchers who looked at the sclerotic patches in the laboratory did not have as stable a patient population, as the French did, to correlate the clinical symptomatology with the pathological laboratory.

Before the Paris Clinic of the first half of the nineteenth-century it would have been unlikely that the sclerotic patches would have been described because of the relatively low frequency of the disease and because of rarity of pathological anatomical studies. The French Revolution led to the reorganization of the Paris hospitals, which greatly centralized and expanded the authority of physicians in the public hospitals. This gave rise to a captive patient population on whom large numbers of autopsies could be performed which resulted in the advances in pathological anatomy of the Paris Clinic. It is no coincidence that what we now consider the first possible illustrations of the sclerotic patches found in multiple sclerosis came out of an institutional matrix that was favorable for advances in pathological anatomy: the Paris Clinic of the 1830s.

J. Cruveilhier's *'Atlas d'Anatomie pathologique'* (Paris 1835-42) and Robert Carswell's *'Pathologic Anatomy'* (London, 1838) are two works from the 1830s who are often cited as the earliest illustrations of the sclerotic patches. Carswell studied with

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[13] Ibid., 633-634.

Pierre Charles Alexandre Louis in Paris during the height of the Paris Clinic in the first third of the nineteenth century. Later he was appointed Chair of Pathologic Anatomy at University College, London. In Carswell's drawing he described,

> a peculiar diseased state of the chord and pons Varolii accompanied with atrophy of the discolored portions, all of them occupying the medullary substance which was hard, semi-transparent, and atrophied.[14]

Cruveilhier was Professor of Pathologic Anatomy in the Faculty of Medicine in Paris during this same period. Contemporaneously with Carswell, Cruveilhier illustrated the sclerotic patches found on the pons and spinal cord of a thirty-one year old woman during autopsy. Cruveilhier included a brief clinical note:

> I followed her during the two years that elapsed from her entry until her death . . . I saw her every week: her intellect was perfect. She smiled at my approach and greeted me with expressiveness. But, when I stopped speaking to her, she would be full of an emotion difficult to describe. She would blush, smile, cry; her limbs and her trunk would be seized with involuntary movements.[15]

In Charcot's " *Leçons sur les Maladies du Système Nerveux faites à la Salpêtrière* (1872-73)," Charcot reported that Carswell made "no reference . . . to any clinical fact." Carswell's drawing was simple a gross illustration of the sclerotic patches. He did not articulate a new disease entity. Charcot described the contribution of Cruveilhier but

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[14] Robert Carswell, *Pathological Anatomy. Illustrations of the Elementary Forms of the Disease*, (London: Longman, Orme, Brown, Green, and Longman, 1838), quoted in Douglas McAlpine, *Multiple Sclerosis* (Edinburgh and London: E. & S. Livingstone, Ltd., 1955), 1.

[15] Translation mine unless otherwise indicated. "Je la suivis pendant les deux ans qui s'écoulèrent depuis son entrée juspqu'a sa mort . . . je la voyais tous les huit jours: son intelligence etait parfaite. Elle souriait à mon approche et me saluait avec expression; mais, quand je lui adressais la parole, elle était prise d'une emotion difficile à rendre. Elle rougissait, riait, pleurait; ses membres et son tronc étaient saisis de mouvement involontaires . . .," J. Cruveilhier, *Anatomie Pathologique du Corps Humain*, volume 2 (Paris: Baillière, 1835-42), 22, quoted in McAlpine, *Multiple Sclerosis*, 1.

added from the 1830s to the 1860s "this question seems almost entirely to have been forgotten." Charcot advised "those who wish to avoid the disenchantment of slow and secondhand discoveries in Pathological Anatomy," to consult Cruveilhier's *Atlas* more often.[16] No where in Charcot's seminal 1868 series of articles on the pathological anatomy of sclerotic patches do the pathological anatomical works of Cruveilhier and Carswell appear. An 1820 work of Cruvelihier, *Dictionnaire de médecíne et de chírurgíe pratiques* (1820) is cited but not the text from the late 1830s considered critical. In other words, there was no linear line of a research tradition from the 1830s to the 1860s. Cruveilhier only described the gross sclerotic patches and a small set of symptoms that would not be identified as signifying a disease or cluster of diseases until the 1870s. Cruveilhier did not construct a new disease entity. It seems that it was only after Charcot's work in 1868 that researchers went back and found the works from the 1830s. The point is that Cruveilhier and Carswell saw only the briefest of glimpses of the sclerotic patches and then this work disappeared from the radar screen. The German pathological anatomists saw glimpses of the sclerotic patches in the 1850s and 1860s but they did not correlate them with clinical observations. The different institutional structure of the *Sâlpetrière* allowed Charcot to combine the clinical and the new laboratory techniques in a way that was difficult in Germany. Still, though Charcot knew that the myelin sheath was being destroyed he did not know why and it would remain debated for decades whether multiple sclerosis was a single disease entity. Whether the condition was primary or secondary of single or multiple etiologies, constitutional or infective was unknown and would be hotly contested in the years to come.

However even if neurologists were not sure what *sclérose en plaques* was in

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[16] J.M Charcot, "Sclerosis in Scattered Patches," in *Leçons sure les Maladies du Système Nerveux faites à la Salpêtrière*, trans. Thomas Oliver in *Edinburgh Medical Journal* 21 (1875-76): 720-721.

terms of a disease in the 1860s, they were beginning to think about what it was not. The difficulty in this condition lay not with the pathologic anatomy at autopsy but with diagnosis during life. It was particularly difficult because neurologists began to realize during the late 1860s and early 1870s that *sclérose en plaques* had a polymorphous clinical presentation. In 1870, neurologist Meredith Clymer wrote:

> In a few cases, as has been stated, the onset is without warning or apparent cause, the patient becoming suddenly paraplegic--in one instance with lessened sensibility. Or the attack may begin with an apoplectic seizure with or without loss of consciousness, and followed by temporary hemiplegia, and fits of vertigo. But in a large majority the approach is insidious, and before any weakness in the lower extremities is complained of, there is tingling in the legs and soles of the feet, with occasional numbness and coldness, and a sense of fatigue after slight exertion. Some stiffness and awkwardness in the movements may be noticed; and in a few instances, probably where the posterior columns of the cord were affected from the outset, there have been darting pain-spells.[17]

In 1868 the French school made the first attempt at a differential diagnosis between paralysis agitans and disseminated sclerosis. In reference to this one author wrote in 1870 that Charcot, "has done for it what Chomel and Louis did for typhoid fever when they established it as a distinct species of continued fever, characterized by a definite group of symptoms."[18] This analogy with the concept of continued fever shows that disseminated sclerosis was being understood in a nosological context that was ***prior*** to the notion of specific diseases with specific etiologies that would arise in the 1880s. It would remain contested for another fifty years whether multiple sclerosis did constitute a

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[17] Meredith Clymer, "Notes on the Physiology and Pathology of the Nervous System, with Reference to Clinical Medicine," New York Medical Journal 11, no. 3 (May 1870): 230, 236. See also, Charcot, "Sclerosis in Scattered Patches (1875-6)," 723. Association for Research in Nervous and Mental Diseases, ***Multiple Sclerosis***, "The Conclusion of the Commission,"(Paul B. Hoeber, 1922), 126. McAlpine, ***Multiple Sclerosis*** (1955), 64-65.

[18] Clymer, "Notes on the Physiology . . ," 231. See also, Charcot, trans. Oliver, "Sclerosis in Scattered Patches, Article VII (1875-76)," 51.

specific primary disease. Others proved less willing or able to diagnose disseminated sclerosis. The French school thought that the English neurologists were particularly confused on this point. Charcot remarked that, "all the English authors confound this disorder with paralysis agitans. Parkinson, whose description of shaking palsy has been closely followed, unquestionably did."[19] The differential diagnosis in the clinic was made possible though not necessarily probable simply by the recognition by the French that paralysis agitans affected the old and disseminated sclerosis seemed to affect young adults.[20]

Even among elite, highly trained neurologists this differential diagnosis was by no means certain. In 1871 leading American neurologist William A. Hammond wrote that "diffused cerebral sclerosis . . . is not a disease which can be recognized with any great degree of certainty or even probability during life." He pointed out that it was often confused with paralysis agitans. Hammond, in contrast to the French researchers, reported that he believed diffused cerebral sclerosis began in infancy and he argued that in the nine cases he had diagnosed all were over fifty years of age.[21]

This diagnostic uncertainty continued in the 1880s. The medical literature on multiple sclerosis in this period is marked by examples, discussions, and debates on how to interpret the mutating, remitting, and variable signs and symptoms of multiple sclerosis in the clinic. Professor Joseph O. Hirschfelder discussed the difficulties the differential

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[19] Ibid., 232.

[20] F.X. Dercum, "Multiple Sclerosis; Traumatic Tremor, Railway Spine," (clinical lecture delivered at the Philadelphia Hospital), *International Clinics: A Quarterly of Clinical Lectures* 1, no. 3 (1893): 122-123.

[21] William A. Hammond, "Diffused Cerebral Sclerosis," *New York Medical Journal* 13, no. 2 (Feb 1871): 129,130. William A. Hammond, "Multiple Cerebral Sclerosis," *The American Practitioner* 3, no. 9 (Mar 1871): 129,139. See also, C.H. Boardman, "Progressive Multiple Cerebro-Spinal Sclerosis," *The Northwestern Medical and Surgical Journal* 3, no. 7 (Jan 1873): 251,256.

diagnosis of multiple sclerosis could entail in a clinical lecture at the Medical College of the Pacific (later to become the Stanford medical school) in 1882. The case he discussed had "produced the signs of tabes dorsalis," which could be easily confused with disseminated sclerosis.

> You can well understand then the anecdote that Charcot relates of a physician, unfamiliar with the disease, who was shown a case. That patient was directed to walk, whereupon the physician remarked that it is a case of tabes. Perhaps, was the answer, but what do you think of the rhythmic motions of the hands and head? Ah! he has likewise chorea or paralysis agitans. The patient was directed to talk, which he did in a scanning manner. I see, answered the physician, you have a very complicated case. Here is a sign of general paralysis. Hold on! your patient seems to unite in himself the whole of nervous pathology.[22]

In 1888 Charcot seemed confident that he could differentiate multiple sclerosis from tabes dorsalis and hysteria but others remained less sanguine for half a century.

At a 1922 New York neurological conference a Dr. Hunt asked: "is there any symptom in multiple sclerosis which one might regard as more or less pathognomonic of the disease--one or more symptoms?" Dr. Bernard Sachs responded:

> . . .There is no one symptom, nor are there any two symptoms that I would consider pathognomonic of the disease. You cannot base a diagnosis of this disease on any one or two symptoms. No doubt many of the old cases of primary or lateral sclerosis come now under the heading of disseminated sclerosis. If you were absolutely to compel me to say which signs have been most helpful to me in making a diagnosis of disseminated sclerosis, I would say that a slight nystagmus, a diminution of abdominal reflex, and a moderate spastic paraplegia-that group of symptoms is to me much more characteristic in the larger number of cases of disseminated sclerosis than is Charcot's triad. We see also many of these cases that have not a trace of speech disturbance or the slightest intention tremor for at least five or ten years during the disease. . . The disease process is so widespread, the number of symptoms that arise is so different in the various cases, that to the best of my knowledge, one should bear in mind

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[22] Joseph Hirschfelder, "Disseminated Sclerosis," *Pacific Medical and Surgical Journal* 25, no. 10 (March 1882)," 446-447.

> at this point at least the first 9 groups of symptoms and try to make the diagnosis, if any number of these are present.[23]

Part of the reason multiple sclerosis was hidden was because of the diagnostic virtuosity required to see it. Landon Carter Gray, Professor of Nervous and Mental Diseases in the New York Polyclinic, reported a confounding case in 1889. Gray treated the man for three years and

> during all this time he presented all the symptoms of a case of disseminated sclerosis, and as such he was observed with interest by my former assistants, Dr. B. Sachs and Dr. M. Allen Starr, and I have also repeatedly lectured upon him as an illustration of this disease.[24]

However upon autopsy no sclerotic patches could be found. This led Gray to diagnosis the cause of death as lepto-meningitis cerebri. In 1922 Walter Timme, M.D. in a review of the literature reported that:

> Of late years, many cases that have conformed in part to the symptomatology of multiple sclerosis have been diagnosed as such only to have the necropsy deny the allegation. American neurologists especially, have been keen to point out clinically that so many cord conditions may simulate multiple sclerosis that many incorrect diagnoses are made and that the correct diagnosis can possibly be made only at the post-mortem examination.[25]

In 1922 it remained uncertain whether multiple sclerosis was one or more diseases,

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[23] ARNMD, Discussion of Sachs and Friedman, "General Symptomatology . . ." Multiple Sclerosis (1922), 57-58.

[24] Landon Carter Gray, "A Case of Lepto-Meningitis Cerebri Presenting Typical Symptoms of Disseminated Sclerosis," *The Journal of Mental and Nervous Disease* 14, no. 2 (Feb 1889): 92-97.

[25] Walter Timme, "Multiple Sclerosis-Historical Retrospect," in Multiple *Sclerosis* (1922), 6.

whether it was a primary condition characterized by the hyperplasia of the glia, or secondary condition following infection or inflammation with subsequent degeneration of the myelin sheaths.[26] This led to a vigorous discussion at the 1922 neurological convention. Following Bernard Sachs presentation, Dr. Carter asked,

> whether or not it is desirable to have a pathological group that we call multiple encephalomyelitis, without going any further, when we have difficulty in deciding whether a patient has multiple sclerosis or spinal lues or encephalitis? Is it not wise, perhaps to have a vaguer syndrome that indicates multiple lesions of which we cannot determine the nature? In other words, make a localizing diagnosis but without definitely deciding as to the nature of the disease?[27]

Dr. Bernard Sachs responded:

> The difficulty, which Dr. Barker has mentioned is rather interesting and has evidently struck European observers. During a visit in Europe last summer, it was interesting to note that in Marie's clinic definite diagnoses are very rarely made. They will speak of the sensory motor complex and the cerebellar complex, and they mention those pathway groupings of symptoms very much more frequently than they mention definite diseases. They are very apt to speak simply of cerebellar groupings, of pontile cerebellar groupings, of opthalmocortical groupings, without in every case making a definite diagnosis.[28]

Sachs admitted that their nosological categories "are constantly in a state of flux." Nevertheless, he thought that in the majority of cases, neurologists had a set of symptoms and signs that reasonably could be held to constitute multiple sclerosis as a specific disease entity. Sach's view seems to have been the minority one at the conference. As biologist Charles B. Davenport put it at the same conference in 1922: "whether we have

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[26] Charles B. Davenport, "Multiple Sclerosis from the Standpoint of Geographic Distribution and Race," in *Multiple Sclerosis* (1922), 14.

[27] Sachs and Friedman, "General Symptomatology of Multiple Sclerosis," in *Multiple Sclerosis* (1922), 60.

[28] Ibid., 62-63.

to do in multiple sclerosis with two diseases, one of which follows an inflammation and degeneration of the nerve sheaths and the other of which is a primary hyperplasia of the glia is uncertain."[29] The 1922 collective judgement of the conference, expressed through the Commission concluded that there was not enough evidence to prove that there was ". . . a clinical and pathological entity such as acute multiple sclerosis," because the multiple sclerosis label might actually cover two or more conditions.[30] Because of the peculiar biology of the phenomenon of the destruction of the myelin sheath and the scarring of nervous tissue and the polymorphous symptoms that ensued this disease took decades to emerge a single pathological entity. Not only was the construct amorphous but its boundaries were as well.

Another nosological category from which disseminated sclerosis migrated and shared porous boundaries from the 1860s to the 1900s was tabes dorsalis, also known as locomotor ataxia, because of the similar symptom of the spastic gait.[31]

> There are several varieties of this form of spinal sclerosis, as one or other column of the cord is chiefly or exclusively affected . . . In such cases the phenomena proper to locomotor-ataxy will be associated with those of the

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[29] Charles B. Davenport, "Multiple Sclerosis From the Standpoint of Geographic Distribution and Race," in *Multiple Sclerosis* (1922), 14.

[30] ARNMD, "Conclusions of the Commission Concerning the Pathology of Multiple Sclerosis," *Multiple Sclerosis* (1922), 208.

[31] The exact etiological relationship between tabes dorsalis or locomotor ataxia and syphilis was not definitively established until the introduction of the Wasserman Test in 1906. For Charcot, syphilis could be a precipitating environmental factor that could trigger a neurological illness in a person with an inherited predisposition. Neurologist William Gowers in 1888 wrote that tabes dorsalis was a degenerative sequel of syphilis rather than a true syphilitic disease. See, Jean-Martin Charcot, *The Clinician The Tuesday Lessons*, trans. with commentary by Christopher G. Goetz (New York: Raven Press, 1987), 18.

spinal form of disseminated sclerosis.[32]

By the 1920s locomotor ataxia usually could be differentiated from multiple sclerosis through the Wasserman Test that was developed in 1906 and the development of the diagnostic technique of Babinski's sign .[33]

The spastic gait and the symptom of scanning speech could also lead to another diagnosis in 1870: alcoholism. "The gait, which from the outset may have been more or less unsteady, is now staggering like that of a drunken man."[34] "The speech is slow, drawling, and now and again almost unintelligible. It seems as if the tongue had become 'too thick,' and the utterance recalls that of people somewhat inebriated."[35] Into the 1950s patients could be diagnosed with chronic alcoholism when they presented the symptoms mentioned above. For example, in 1956 the hand of a young man in his thirties suddenly went numb and he found he would drop his cigarettes. His handwriting also deteriorated. He was diagnosed as a chronic alcoholic. Later he would be diagnosed

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[32] Clymer, "Notes on the Physiology . . . ," 235. See also, C.H. Boardman, "Progressive Multiple Cerebro-Spinal Sclerosis," *The Northwestern Medical and Surgical Journal* 3, no. 7 (January 1873): 251. Charcot, trans. Oliver, "Sclerosis in Scattered Patches, Article VII (1875-76)," 55. C.H. Hughes, "Inability to Gargle and Inco-Ordinate Backward Movements, Additional Signs of Cerebral and Posterior Spinal Sclerosis," *New York Medical Journal* (25 Apr 1885): 472. L.C. Gray, "A Case of Lepto-Meningitis Cerebri Presenting Typical Symptoms of Disseminated Sclerosis," *Journal of Nervous and Mental Diseases* 16 (1889)," 92-98.

[33] Sachs and Friedman, "The Differential Diagnosis," 133-136.

[34] Clymer, "Notes on the Physiology . . . ," 234.

[35] Charcot, trans. Oliver, "Sclerosis in Scattered Patches, Article V (Aug 1876)," 118. See also, E.C. Seguin, "A Contribution to the Pathological Anatomy of Disseminated Cerebro-Spinal Sclerosis," *Journal of Nervous and Mental Diseases*, 5 (1878): 282-283.

as a victim of multiple sclerosis.[36]

Probably the largest reinterpretation of the presenting symptoms of disseminated sclerosis and the resulting nosological trans-formation occurred between the categories of hysteria and multiple sclerosis.[37] Many elite neurologists struggled with the difficulties in reading the differential symptoms. For example, Dr. E.C. Seguin, Clinical Professor of Diseases of the Mind and Nervous System, College of Physicians and Surgeons, Columbia University, gave a case history before the New York Neurological Society in February 1878 of a single, twenty-three year old woman first seen on 20 October 1873:

> A nervous girl, with occasional irregularity of menstruation, but no dysmenorrhoea. At times hysterical laughter and tears; never convulsive attack. In July, 1871, while out walking, after having climbed a number of walls, felt weak and awkward in right leg; thought she had sprained her knee. There is not enough evidence to support this statement. Ever since she has had weak right leg, without anaesthesia or numbness; at time more use of leg than at others; almost cured once or twice; of late has required help of crutch, or friend's arm in walking. When I examined Miss P. I found paresis of right leg, the loss of power being marked at ankle and toes. There was doubtful weakness of the right hand. I could not make out if the knee joint was affected. The muscles of the right leg showed a slight diminution of reaction to the faradic current, and this agent also showed that sensibility to pain was a little dull in leg and foot. In view of the history of the case, the capricious development of the palsy, the absence of reliable signs of central disease, the presence of a strong neurotic element in the family, and the fact the strong emotions had been

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[36] University of California, Los Angeles, Special Collections, *Tracy Jackson Putnam M.D. Collection, 1938-1975, Ms Coll. #90* (Hereafter *Putnam Archives*), Paul Teng, Long Beach, CA to Tracy J. Putnam, Beverly Hills, CA, (7 August 1957), Letter, *Putnam Archives*, Box: 2, Folder: An, My.

[37] Mark S. Micale argues that hysteria did not disappear so much as migrate into a multitude of new nosological categories after the late nineteenth century. The most important of these categories included: syphilis, epilepsy, various German psychotic categories and Freudian psychoneuroses. Micale only mentions multiple sclerosis in passing. The problem with this argument is that if hysteria is a reified concept, as Micale argues, then how can what does not really exist migrate? Perhaps it is better say that these various clinical symptoms and signs had a wide range of textual possibilities and could be read in a number of ways depending on the social or cultural context. See Mark S. Micale, "On the 'Disappearance' of Hysteria: A Study in the Clinical Deconstruction of a Diagnosis," *Isis* 84 (1993): 496-526.

> acting upon her, I concluded that the patient had a functional palsy of an hysterical nature.[38]

The patient died on 1 August 1874 and the autopsy revealed "disseminated sclerosis of the spinal cord." For Charcot in 1888 the central clinical problem in *sclérose en plaques* remained how to differentiate it from *ataxie locomotrice* and *l'hystérie*.[39] Still, in 1922, American neurologists had to be urged that it was "particularly important not to confound the early cases of multiple sclerosis with hysteria and treat them as such."[40] However, because multiple sclerosis was marked by unusual remissions and improvement, it "not infrequently" continued to be diagnosed as hysteria in the 1920s.[41] Hysteria would still be diagnosed in cases that were later considered multiple sclerosis throughout the 1950s.[42] For example, in 1950 a young woman temporarily went partially deaf and was diagnosed by a psychiatrist with hysteria. In 1954 she developed numbness and weakness in her left hand and this too was coded hysterical. In 1955 she suddenly fainted as she got out of bed. Following this she was described as giddy and had difficulty walking down stairs. The diagnosis was as follows:

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[38] Seguin, "A Contribution to the Pathological Anatomy of Disseminated Cerebro-Spinal Sclerosis (1878)," 285-6.

[39] Jean Martin Charcot, "*Policlinique du Mardi 11 Décembre 1888, Huitième Leçon*," in *Leçons du Mardi a la Salpêtrière*, ed. E. Lecronier and Babé (Paris: Progrès Médical, 1888-1889), 22, 162-171.

[40] Bernard Sachs and Emanuel D. Friedman, "The Differential Diagnosis, Course and Treatment of Multiple Sclerosis," *Multiple Sclerosis* (1922), 142.

[41] Bernard Sachs and Emanuel D. Friedman, "General Symptomatology of Multiple Sclerosis," *Multiple Sclerosis* (1922): 53, 58, 59. Sachs and Friedman, "The Differential Diagnosis," 141.

[42] *Putnam Archives*, Box: 5, Folder: Co, Gr, "Patient Exam Record" (16 February 1951), 1. For ten days the patient temporarily went blind two years before seeing Putnam. At that time her psychiatrist diagnosed it as a form of hysteria.

> Her personal history is highlighted by constant clashes with her 'strict, oppressive mother' which caused her to go to New York in revolt. While she apparently had no trouble in finding and holding jobs, making friends, she continued to depend on her folks and she made several trips to Los Angeles on visits. She had two years of psychoanalysis after 1950--with some intellectual insight but without protection against various nervous upsets. She is under psychotherapy at present . . . I believe that this patient has the labile autonomous nervous system of hysteroid personalities which causes her to be an easy candidate to vasospasms.
>
> There is nothing to contradict the diagnosis of conversion hysteria and she is getting the treatment for it.[43]

A neurologist later diagnosed her condition as multiple sclerosis.[44]

This tendency to code the polymorphous symptoms involved in multiple sclerosis as hysterical was rooted in cultural assumptions about gender. Between the 1890 and 1920s, for example, the orthodox medical view in Anglo-American circles was that men were more affected than women with multiple sclerosis by a ratio of 3 : 2, with a similar ratio for the United States and Europe.[45] A large 1950 study in the United States reported an incidence of 42.5% for men and 57.5% for women, or a changed ratio of men to women of 1.4 : 2. And, in the United States in the 1990s women are considered twice as likely to suffer from multiple sclerosis as men.[46] [See Tables V and VI.] What accounts for this epidemiological shift over time?

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[43] Nicholas A. Bercel, M.D., Beverly Hills, CA, to Julius Ziegler, M.D., Los Angeles California, (12 September 1955) Letter, ***Putnam Archives***, Box: 10, Folder: Gu, Th.

[44] Tracy Jackson Putnam, M.D., Beverly Hills, CA to Ziegler, Julius, M.D. Los Angeles, CA (26 Sept 1956), Letter, ***Putnam Archives***, Box:10, Folder: Gu, Th.

[45] Israel S. Wechsler, "Statistics of Multiple Sclerosis," in ***Multiple Sclerosis*** (1922), 34. See also, Smyth Ely Jelliffe, "Multiple Sclerosis: Its Occurrence and Etiology," ***Journal of Nervous and Mental Diseases*** 31 (1904): 450.

[46] Lawrence Steinman, "Autoimmune Disease," ***Scientific American*** (Sept 1993): 107.

Throughout the nineteenth-century in Europe and North America, hysteria was considered the most common neurological condition that affected women between menarche and menopause.[47] In 1816 the French alienist J.B. Louyer-Villermay wrote: "Man cannot be hysterical . . . He has no uterus."[48] Hysteria in males was considered rare and when it was diagnosed the effeminate nature of the hysterical males would often be noted. In 1845 Viennese alienist Ernst von Feuchtersleben wrote that, "when men are attacked by genuine hysterical fits . . . which certainly does occur, they are, for the most part effeminate men."[49] John Russell Reynolds in, *A System of Medicine* (1866-79), argued that "when hysteria is found in either a man or a boy, it is to be observed that such person is, either mentally or morally, of feminine constitution."[50] In 1891, Gilles de la Tourette fixed hysterogenic points on the hysteric male body that he called the pseudo-ovarian zone.[51] It was this received orthodoxy that Charcot attempted to revise in 1889 with the publication of his studies on male hysteria. Charcot did not deny that effeminate men could become hysteric "just like a women" but manly and vigorous artisans like

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[47] Mark S. Micale, "Charcot and the Idea of Hysteria in the Male: Gender, Mental Science, and Medical Diagnosis in Late Nineteenth-Century France," *Medical History* 34 (1990): 370.

[48] J.B. Louyer-Villermay, *Traité des maladies nerveuses ou vapeurs et particulièrment de l'hysterie et de , Volume 1* (Paris: J.B. Baillière, 1816), 116, quoted in Micale, "Charcot and the Idea of Hysteria in the Male," 368.

[49] Ernst von Feuchtersleben, *The Principles of Medical Psychology*, trans. H.E. Lloyd, rev. and ed. B.G. Babington (London: Sydenham Society, 1847), 228, quoted in Micale, "Charcot and the idea of hysteria in the male," 380.

[50] J. Russell Reynolds, "Hysteria," in ed. Reynolds, *A System of Medicine*, volume 2 (London: Macmillan, 1866-79), 307, quoted in Micale, ibid., 380.

[51] Gilles de la Tourette, *Traité clinique et thérapeutique de l'hystérie d'après l'enseignment de la Sâlpêtrière, volume 1* (Paris: Plon, Nourrit, & Cie, 1891), 299-300, quoted in Micale, ibid., 403-404.

train engineers could also become hysterical. However, the reasons were different: Charcot tried to show that while an excess of feminine behaviors such as marital discord, unrequited love, religious mania, fear, and grief could lead women into hysteria, vigorous artisan men became hysteric through an excess of masculine behaviors such as: working, drinking, or fornicating too much.[52] A certain ambivalence permeated Charcot's case studies. The working-class men that constituted the bulk of Charcot's case studies had often failed in fights or at work. In the *mentalité* of the late-nineteenth century working-class not being able to work would have been seen as a dependent feminizing state.[53] As a result, neurologists were reluctant to label men as hysteric during the late nineteenth-century because of the pejorative connotations that diagnosis carried.[54] Though Charcot argued that male hysteria was more common than previous authors had believed, he still estimated the ratio of hysteric women to men to be 20:1.[55] Let us look at several case histories to see how the cultural lens of gender filtered the wide textual possibilities of the pathognomic signs in multiple sclerosis.

English neurologist Thomas Buzzard presented the case of a thirty-three year old male house painter in a clinical lecture on 9 April 1875 to illustrate the symptomatology of disseminated sclerosis.

> Asked to rise, the patient . . . made at first several ineffectual efforts, his whole body being thrown into violent tremors, the feet being lifted from

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[52] Micale, "Charcot and the Idea of Hysteria in the Male," 406.

[53] See S. Maynard, "Rough Work and Rugged Men--The Social Construction of Masculinity in Working-Class History," *Labour-Travail* 23 (Spring 1989): 159-169. J.E. Smith, Gender and Class in Working-Class History," *Radical History Review* 44 (Spring 1989): 152-158.

[54] Micale, "Charcot and the Idea of Hysteria in the Male," 377-379.

[55] Ibid., 406.

> the ground . . . Then he began to have attacks of giddiness . . . There was some deficiency of power in all four limbs . . . The sight was somewhat enfeebled . . . He could not read without embarrassment.[56]

If this had been a woman it is likely she would have been diagnosed with hysteria. Note the gendered phrasing and tone of the clinical description. It is hardly indicative of Charcot's manly form of hysteria. Joseph O. Hirschfelder, Professor of Clinical Medicine at Medical College of the Pacific, presented a sixty-seven year-old Irish tailor as an example of disseminated sclerosis in 1882: "for twenty years he has been subject to vertigo . . . His power of thought has diminished, and he frequently cries without provocation."[57]

Contrast these cases with this twenty-three year old female patient seen by E.C. Seguin, Clinical Professor of Diseases of the Mind and Nervous System, College of Physicians and Surgeons, on 20 October 1873.

> A nervous girl, with occasional irregularity of menstruation . . . At times hysterical laughter with tears . . . In July 1871, while out walking, after having climbed a number of walls, felt weak and awkward in right leg . . . Sensibility to pain was little dull in leg and foot.

Seguin diagnosed her as hysteric but on postmortem examination disseminated sclerosis of the spinal cord was discovered.[58] Because of the gendered filter through which neurologists read and interpreted the symptoms, signs, and symbols generated during the clinical encounter between the physician and patient men were much more likely to be

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[56] Thomas Buzzard, "A Case of Disseminated Sclerosis, with Special Reference to the Frequency and Etiology of the Disease," *Clinical Studies, A Quarterly Journal of Clinical Medicine* 2 (1904): 121-124.

[57] Hirschfelder, "Disseminated Sclerosis," 434-439.

[58] Seguin, "A Contribution to the Pathological Anatomy of Disseminated Cerebro-Spinal Sclerosis," 285-286.

diagnosed with multiple sclerosis than were women even if they had identical symptoms. Consider these pathognomic signs in an example from, *The Diseases of the Spinal Cord* (1882), by Byrom Bramwell, Professor of Medicine, University of Edinburgh:

> The patient, a chemical worker, æt 28, was admitted to the Newcastle-on-Tyne Infirmary, under my care, on September 10th, 1874, complaining of, difficulty in walking, loss of power in the lower extremities, pain in the back, and giddiness. His illness had commenced six years previously, and was apparently due to a severe blow on the back of the head. He had been a very heavy drinker. He was a very large muscular man. There was no apparent loss of motor power. His movements were decidedly unsteady: the incoordination was, however, quite different from the inco-ordination of locomotor ataxia; it was, so to speak, of a 'coarser' kind and involved the muscles of the trunk rather than the muscles of the lower extremities. In walking, the neck was held stiff, the head turned slightly to one side, and the eyes raised from the ground. On close observation, a fine jerking rhythmical movement of the head could be observed. There was also occasional nystagmus. His memory was defective; he was very irritable and easily excited. He complained every now and again of double vision, but nothing abnormal could be detected in connection with the muscles of the eyeball, or with the fundus. The speech had undergone marked alteration during the past six months; and at the time of his admission, was thick and drawling. Pain in the back was at this time a prominent symptom; it was referred to the lower dorsal region, and the patient complained of tenderness on percussion over the same part.[59]

This is an example of the polymorphous clinical presentation of multiple sclerosis. The clinician had many diagnostic possibilities as he read the text of the clinical signs. One possible reading would have been to link the patient's giddiness, defective memory, irritability, and excitability into a diagnosis of hysteria. But, since the patient was a "very large muscular man" this reading would have been unlikely. The point is that similar clinical signs were often read in different ways depending on the sex of the subject.

The same Dr. Byrom Bramwell writing in 1904 concluded: "I would almost go so far as to say that the great majority of cases of disseminated sclerosis, in women, are, in

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[59] Byrom Bramwell, *The Diseases of the Spinal Cord* (New York: William Wood & Company, 1882), 56-57.

their early stages, thought to be cases of hysteria."[60] As the diagnosis of hysteria declined and dispersed after its apogee in the late nineteenth-century, many of those who might have been categorized as female hysterics based on a constellation of pathognomic signs gradually migrated to the nosological category of multiple sclerosis throughout the twentieth-century.

The persistence of the medical mystery and difficulties of multiple sclerosis in terms of nosology, etiology, and diagnosis meant that the framing of multiple sclerosis ought not to be characterized as a discovery event but rather as a medical *uncovering* which was a slow process that continued from the late 1860s into the 1950s. The peculiar biology of multiple sclerosis linked it early on to tertiary syphilis and hysteria nosologically and morally. This served to stigmatize the disease by association and is one reason the disease remained out of sight for so long. This stigmatization also occurred because of *fin de siècle* assumptions about degenerative neurological diseases.

After Charcot's work in the 1860s, many neurologists thought *sclérose en plaques* to be a diathetic condition or an organic endogenous inherited disease. The French degenerationist theories reached their peak in the 1880s. In the positivistic materialistic neurological thought of the late nineteenth-century, most nervous disorders including the conditions and behaviors: alcoholism, suicide, criminality, insanity, and homosexuality were thought to be somatically based.[61] Irwin H. Neff and Theophil Klingmann in, "A Case of Multiple Cerebro-Spinal Sclerosis of a Special Anatomical Form, with a History of Pronounced Family Defect," in the *American Journal of Insanity* (1899) suggested that

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[60] Byrom Bramwell, "On Disseminated Sclerosis, with Special Reference to the Frequency and Etiology of the Disease," *Clinical Studies, A Quarterly Journal of Clinical Medicine* 2 (1904): 193-194.

[61] Jean-Martin Charcot, *The Clinician The Tuesday Lessons*, trans. with commentary by Christopher G. Goetz (New York: Raven Press, 1987), 5, 8, 18.

multiple sclerosis was a condition that might occur in a family with other neurological "degenerations" including: chronic nervous trouble, insanity, meningitis, spina bifida, hysteria, epilepsy, and consumption. [See Table VII.] They maintained that,

> mental defect has been noticed since the patient was 13 years of age . . .
> No history of any infective trouble could be obtained . . .
> Anthropometrical measurements and the examinations show the . . .
> physical stigmata of degeneration.[62]

British physician Ernest S. Reynolds, writing in *Brain* (1904) maintained that in multiple sclerosis it was rare to find instances of direct heredity that ran in families. However, Reynolds following neurologist William Gowers believed that "indirect inheritance is more commonly, shown by a family history of insanity, epilepsy, or of some form of chronic paralysis."[63] Even though little evidence had accumulated for direct inheritance from parents or grandparents, Reynolds argued:

> I am inclined to think, however, that the more we become familiar with the aberrant types of disseminated sclerosis, the more it may be found to occur in families; that is to say, where one member of a family has typical disseminated sclerosis, another member who has been diagnosed to be merely 'neurotic' may really be found to be suffering from aberrant disseminated sclerosis."[64]

This was within accepted neurological thinking of the period and within eugenic thinking as well. In a presentation before the Association of Assistant Physicians of Hospitals for the Insane, at Cleveland, Ohio, September 26-28, 1899, Drs. Irwin H. Neff and Theophil

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[62] Irwin H. Neff and Theophil Klingmann in, "A Case of Multiple Cerbro-Spinal Sclerosis of a Special Anatomical Form, with a History of Pronounced Family Defect," *American Journal of Insanity* 56 (1899): 433.

[63] Ernest S. Reynolds, "Some Cases of Family Disseminated Sclerosis," *Brain* 27 (1904): 167.

[64] Ibid., 168.

Klingman presented the case of a family with a pronounced degenerate "defect." A 52 year-old man diagnosed with multiple sclerosis was hereditarily tainted. As evidence the authors produced a family tree that linked through genetic causation multiple sclerosis, epilepsy, chronic nervous trouble, cancer, consumption, meningitis, puerperal fever, spina bifida, hysteria, deafness, and imbecility.[65] Since multiple sclerotics were guilty by association the disease was stigmatized and kept largely hidden.

A. Meyerson, in a paper read at the Second International Congress of Eugenics, held at the American Museum of Natural History in New York City from September 22-28, 1921, argued that, "there is very much general neuropathic heredity in the direct and collateral relatives of both the insane and sane."[66] Paul Popenoe, editor of the *Journal of Heredity* and Roswell Hill Johnson, a professor at the University of Pittsburgh, in a chapter entitled, "Dysgenic Classes," in their *Applied Eugenics* (1918), maintained that:

> In addition to these well-recognized classes of hopelessly defective, there is a class of defectives embracing very diverse characteristics, which demands useful consideration. In it are those who are physical weaklings or deformed, those born with a hereditary diathesis or predisposition toward some serious disease (e.g. Huntington's Chorea), and those with some gross defect of the organs of special sense . . . In many cases the affected individual himself will be glad to cooperate with society by remaining celibate or by the practice of birth control, to the end of leaving no offspring to bear what he has borne."[67]

Michael F. Guyer, Professor of Zoology, University of Wisconsin, in Being Well-Born: and Introduction to Heredity and Eugenics (1st edition 1916, second edition 1927) wrote

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[65] Irwin H. Neff and Theophil Klingmann, "A Case of Multiple Cerebro-Spinal Sclerosis of a Special Anatomical Form, with a History of Pronounced Family Defect," *American Journal of Insanity* 56 (1899): 431-433.

[66] A. Meyerson, "Inheritance of Mental Disease," in *Eugenics, Genetics and the Family, Volume 1* (Baltimore: Williams and Wilkins Co., 1923), 219.

[67] Paul Popenoe and Roswell Hill Johnson, *Applied Eugenics* (New York: The MacMillan Company, 1918), 180.

that:

> The brain mechanism is as much a product of ancestry as in any other structure of the body, and it is obvious therefore that imperfect adjustments of its structure must be as subject to the laws of inheritance as other malformations of the body. Because of the extreme complexity and delicacy of its mechanism, it is peculiarly liable to derangements which even when slight, may have fare-reaching effects. [68]

Samuel J. Holmes, Professor of Zoology, University of California, Berkeley, in The Trend of the Race: A Study of Present Tendencies in the Biological Development of Civilized Mankind (1921) concluded that:

> Since Morel published his celebrated treatise on Degeneracy in 1857, it has been a prevalent idea that many forms of defect and disorder are not transmitted as such, but may give place in the descendants to abnormalities of the most varied kind. What is transmitted is held to be a degenerate constitution which may be manifested in diverse ways according to circumstances."[69]

Philadelphia physician Alfred Gordon writing in the Eugenical News in 1935 argued that with respect to MS, "we are therefore dealing here with some congenital defects of the nervous system transmitted to some descendant in the form of Disseminated Sclerosis."[70] In the scientific culture of the early twentieth-century it was not unusual that multiple sclerosis was considered an inherited degenerate condition. What was unusual was who was being diagnosed with the disease.

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[68] Michael F. Guyer, *Being Well-Born: an Introduction to Heredity and Eugenics* (New York: Bobbs-Merrill Company, 1927, original 1916), 339.

[69] Samuel J. Holmes, *The Trend of the Race: A Study of Present Tendencies in the Biological Development of Civilized Mankind* (New York: Harcourt, Brace, and Company, 1921), 64.

[70] Alfred Gordon, "The Problems of Heredity and Eugenics," *Eugencial News* 20, no. 4 (July-August 1935): 52.

III

WHO GETS IT AND WHY?

Scientific and cultural assumptions about who should be suffering from neurological "degeneration" conflicted with the peculiar epidemiological portrait of MS. The direction laboratory research concerning MS took in the 1910s and 1920s, toward the infective hypothesis, was possible because the continuing deep mystery of its etiology gave room for theoretical speculation in the laboratory to explore possibilities beyond what was paradigmatic in terms of disorders of neurological "degeneration." Specifically, when most neurological disorders of the day were considered inherited, research on multiple sclerosis turned toward infection, forty years after the germ theory and the doctrine of specific etiology emerged. The question is why this research occurred specifically in the 1910s and 1920s. I hope to suggest that it was because of the larger cultural conceptions embedded in the eugenics movement.

Walter Timme, M.D., writing in 1922, said that, "only in very recent years has the view that multiple sclerosis is a disease *per se* begun to obtain more and more supporters."[71] American neurologist Smyth Ely Jelliffe writing in 1904 concluded that:

> trauma, poisoning, acute infectious diseases, any cause lowering the power of an hereditary endowment, any abiotrophy, have each in turn been assumed to play a dominant role in multiple sclerosis.[72]

Besides these, causes might include: insanity, grief, mental shock, emotional upset, fatigue, surgery, syphilis, parturition, excess, chills. Though the doctrine of specific

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[71] Timme, "Multiple Sclerosis-Historical Retrospect," in *Multiple Sclerosis* (1922) 7.

[72] Smyth Ely Jelliffe, "Multiple Sclerosis its Occurrence and Etiology," *Journal of Nervous and Mental Diseases* 31 (1904): 453.

etiology became paradigmatic after the 1890s it was not applied to multiple sclerosis in any clear way because it remained unclear in the minds of neurologists if multiple sclerosis was a primary disease. This is not difficult to understand considering the vast array of what were considered precipitating events. In the 1890s French neurologist Marie argued that an infectious or toxic agent could be the precipitating cause of *sclérose en plaques* . This was not really in opposition to the *Salpètrian* school because it meant only people of degenerate hereditary stock would be affected by the unknown environmental agent or agents. [73] Indeed this is precisely the argument put forward by the leading eugenic scientist in the United States, Charles B. Davenport at the 1922 neurological conference. Davenport argued that geographical, ethnological, and familial distribution of multiple sclerosis depended in part on one or more hereditary factors. If an endogenous agent was also involved the factor of heredity would still be important. Davenport argued that:

> Just as tumors inoculated into a mouse will, or will not, grow, according to the racial constitution of the mouse; and just as the bacillus tuberculosis that inhabits the body of all of us does or does not flourish there depending upon the constitution and condition of the individual; so there are probable internal conditions that inhibit, and others that facilitate the development of this disease or the endogenous factors upon which it depends."[74]

The key question in medical research communities was: if it were a primary disease what kind would it be considering the anecdotal evidence of the first decade of the twentieth-century and harder epidemiology evidence from the second decade of the twentieth-century seemed to suggest that it seemed to affect northern middle and upper

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[73] Ibid., 453-454.

[74] Charles B. Davenport, "Multiple Sclerosis from the Standpoint of Geographic Distribution and Race," *Multiple Sclerosis* (1922), 18-19.

whites more frequently. [75] Scottish neurologist Byrom Bramwell writing in 1904 reported: "in my hospital practice, the disease is more common in the well-to-do (hospital) patients than in the very poor."[76] [See Table VIII.] Statistical analyses at that time had established the usual age of onset from the late teens through the early thirties. By World War I, partly through statistics garnered during massive medical examinations of drafted soldiers, it became clear there was a geographic pattern to multiple sclerosis. The northern latitudes of the United States and of the north of England and of Europe, especially the Nordic populations, had much higher proportions of multiple sclerosis than did more the southerly regions. In terms of ethnicity in the United States people of English, Scottish, German, Swedish, and Norwegian stock had a much higher incidence of multiple sclerosis than Russians, Eastern Europeans, Italians, and Irish. In terms of race, African-Americans, Asians, and American Indians had the lowest incidence. As was reported in the American popular press in 1930, "strangely, blond, blue-eyed people

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[75] Byrom Bramwell, "The Relative Frequency of Disseminated Sclerosis in this Country (Scotland and the North of England) and in America," *Review of Neurology and Psychiatry* 1 (1903): 12-17. This Scottish neurologist, on comparing his statistics in Scotland and the north of England with those from New York City, concluded that disseminated sclerosis was three and one-half times greater in Britain than in the United States. Of course he was comparing the zone of highest concentration in Britain with a very limited American sample. Bramwell was aware of the problematic nature of the evidence. See, Byrom Bramwell, "On Disseminated Sclerosis, with Special Reference to the Frequency and Etiology of the Disease," *Clinical Studies, Edinburgh* 2 (1903-04): 193-210. R.M. Van Wart, "A Note on the Frequency of Multiple Sclerosis in Louisiana," *New Orleans Medical and Surgical Journal* 57 (1904-05): 549-551. S.E. Jelliffe, "Multiple Sclerosis: Its Occurrence and Etiology," *Journal of Nervous and Mental Diseases* 31 (1904): 446-455. L.H. Mettler, "Case of Disseminated Cerebrospinal Sclerosis with a Suggestive Family History," *Chicago Medical Recorder* 27 (1905): 290-293. I.H. Neff and T. Klingmann, "A Case of Multiple Cerebro-spinal Sclerosis of a Special Anatomical Form, with a history of pronounced family defect," *American Journal of Insanity* 56 (1899-1900): 431-442. E.S. Reynolds, "Some cases of family disseminated sclerosis," *Brain* 27 (1904): 163-169.

[76] Bramwell, "On Disseminated Sclerosis, with Special Reference to the Frequency and Etiology of the Disease," 200.

seem to be most susceptible," to multiple sclerosis.[77] [See Tables IX and X.] This knowledge of the ethnic and racial distribution of multiple sclerosis emerged during the height of the eugenics movement in the United States and Britain from the 1900s-1930s. As I said Charles B. Davenport himself presented the statistics on the geographic and racial distribution at the 1922 neurological conference. Clearly neurological and eugenical thought were at least co-existing in the same discursive space. Here was a believed inherited degenerate neurological condition of white, northern Europeans and Euro-Americans who were as likely to be affected in the more virtuous rural areas as they were in the neurasthenic cities regardless of class.[78] These were the wrong people to be suffering from tainted heredity. What impact did this finding have on the scientific beliefs in the context of the larger assumptions in neurology, psychiatry, biology, and middle and upper-class Anglo-Saxon culture about race and ethnicity?

That context is exemplified by the work of the prominent biologist and eugenicist Charles Davenport who garnered ten million dollars from the Carnegie Foundation to set up the Cold Spring Harbor research laboratory in 1904. Davenport began work on the relationship between eugenics using the tools of the rediscovered Mendelian genetics. He published *Heredity in Relation to Genetics* in 1911. Davenport argued that hemophilia, otosclerosis, Huntington's chorea, insanity, epilepsy, alcoholism, pauperism, criminality, feeblemindedness, and multiple sclerosis were heritable.[79] Daniel J. Kevles argues that,

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[77] "The Tiniest Germ: Organism Responsible for Creeping Paralysis," *Literary Digest* 106 (30 Aug 1930): 29. Watson Davis, "Development of the Ultra Microscope," *Current History* 32 (Sept 1930): 1170.

[78] Jelliffe, "Multiple Sclerosis . . . (1904)," 451-452. Bramwell, "On Disseminated Sclerosis . . . (1903-04)" 209.

[79] Daniel J. Kevles, *In the Name of Eugenics: Genetics and the Uses of Human Heredity* (Berkeley: University of California Press, 1985), 46, 56.

> like Galton and Pearson, Davenport identified good human stock with the middle class--especially 'intellectuals,' artists and musicians, and scientists. In his American context, he also gave high marks to the native white Protestant majority.[80]

Davenport became primarily interested in negative eugenics: the prevention of the breeding of "bad stock." Davenport secured $500,000 from the Carnegie Foundation between 1910 and 1918 with another $22,000 from the Rockefeller Foundation for eugenics research. He deployed two hundred and fifty eugenics field worker between 1911 and 1924 to gather data. In 1916 his office began publishing the *Eugenical News* which gave an authoritative scientific voice to the questions of sterilization of the unfit and the heritability of various diseases including multiple sclerosis.[81] By 1914 thirty states had restrictive marriage laws based on eugenical principles and by 1917 sterilization laws were widespread in the United States. In ***Buck v. Bell*** (1927) the United States Supreme Court found the Virginia sterilization law constitutional. Justice Oliver Wendell Holmes, Jr wrote the decision. He stated:

> We have seen more than once that the public welfare may call upon the best citizens for their lives. It would be strange if it could not call upon those who already sap the strength of the state for these lesser sacrifices . . . Three generations of imbeciles are enough.[82]

By 1929, 6,255 sterilizations had been performed in California alone. From the early 1920s to the late 1930s the rate of sterilizations in the United States increased from 2 per

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[80] Ibid., 47.

[81] Ibid., 55-56.

[82] Quoted in, Stephen Jay Gould, *The Mismeasure of Man* (New York: W.W. Norton & Company, 1981), 335.

100,000 to 20 per 100,000.[83] To reiterate an earlier point this sort of negative association of multiple sclerosis with tainted heredity and other degenerate neuropathic disorders would have served to stigmatize multiple sclerosis and keep it off of the cultural radar screen especially considering the wide popularity of eugenic ideas and eugenic marriage manuals among the white middle and upper classes in the United States.

Similar ideas about the hereditarian roots of nervous disorders were current in Britain. However disputes between the Mendelians and biometricians divided the British geneticists eroding the scientific authority with which British eugenicists could proselytize. The Germans were even more enthusiastic than the Americans in their embrace of eugenics and theories of degeneration and heredity. In the Third Reich this would be taken to its criminal extreme.[84] This was the context in which the findings that Scandanivians, Germans, and British stock seemed to have a higher incidence of multiple sclerosis emerged. Others remained convinced, regardless of the results to the contrary that multiple sclerosis was an infectious disease.

For example the eugenicist and neurologist Charles L. Dana, Professor of Neurology, Cornell University Medical College, at the 1922 MS conference wrote that "I am assuming that there is an infecting organism at the bottom of multiple sclerosis lesions. I even assume it is an animal type, i.e., some variety of spirochaetae; hence I place the problem of multiple sclerosis in the domain of animal ecology," and, thus, not in the domain of tainted heredity.[85] Why would Dana seem to be arguing against the

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[83] Kevles, *In the Name of Eugenics*, 115-116.

[84] Ibid., 105.

[85] Charles L. Dana, M.D., "Multiple Sclerosis and the Methods of Ecology,"*Multiple Sclerosis* (1922), 43. Dr. Charles L. Dana is listed as a member of the Committee on Cooperation with Physicians of the American Eugenics Society in American Eugenics Society, Inc., *Organized Eugenics* (New Haven, January 1931), 24.

dominant eugenic and neurological thinking? Dana supervised a Dr. Mary M . Sturgis, who had trained with Charles Davenport, as she studied 10 cases of multiple sclerosis. Sturgis, "visited the homes of these patients, seen them and their families and obtained details of the life of patient and family and the incidence and development of the disease." Dana concluded from the study that, "it seems to me that multiple sclerosis is relatively rare in the tuberculous, and I cannot find it in the mental defectives, nor much of it in the strictly laboring classes, or muscle workers."[86] The problem was the wrong sort of people seemed to have multiple sclerosis with regards to the eugenical assumptions about degenerative nervous diseases. Dana rejected the tainted heredity argument not based on the evidence but based on his prior assumptions.

Let's look at the dubious bacteriological studies from 1913 to 1921 to see on what other scientific basis Dana might have made his judgement. In 1913 British researcher W. E. Bullock injected the spinal fluid of a multiple sclerosis patient into several rabbits and one cat. Because several of the rabbits became paralyzed, Bullock concluded that multiple sclerosis was "a disease due to a filter passing organism, or to a water soluble poison."[87] In 1917 German scientists P. Kuhn and G. Steiner injected guinea-pigs and rabbits with the spinal fluid, blood, and blood mixed with spinal fluid of multiple sclerosis patients. They asserted they had passed the paralytic disease through four passages in guinea-pigs and two passages in rabbits. They concluded the disease was caused by a spirochete similar to the leptospira of Wiel's disease (infective jaundice)

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[86] Dana, "Multiple Sclerosis and the Methods of Ecology," 45-46.

[87] Oscar Teague, "Bacteriological Investigation of Multiple Sclerosis," *Multiple Sclerosis* (1922), 121-122. W. E. Bullock, "The experimental transmission of disseminated sclerosis to rabbits," *Lancet* 2 (1913): 1185.

which they named *Spirochaeta argentinesis*.[88] In 1918 French researchers Simons and G. Marinesco said they had confirmed the findings of Guy, Kuhn, and Steiner.[89]

In 1921 Rothfeld, Freund, and Hornowski injected the blood, spinal fluid, and organ emulsions of multiple sclerosis patients into guinea-pigs and rabbits. They had completely negative bacteriological results and were thus unable to confirm Kuhn's and Steiner's research. Many of their animals died from coccidiosis, pulmonary inflammation, cellular infiltration of the kidney, and tuberculosis.[90] That same year Magnus in Christiana used similar techniques but could not produce paralysis in his animals. W.E. Bullock (Guy) attempted a more extended control of her original study. This time, of the 15 guinea-pigs and 129 rabbits, all the guinea-pigs survived, 112 of the rabbits survived, and 17 of the rabbits became paralyzed. Guy detected an extensive coccidiosis among several of the dead rabbits. Nevertheless, she concluded that multiple sclerosis was probably an infectious disease caused by a virus which occasionally could be identified in the spinal fluid.[91] In 1922 the Commonwealth Foundation made a grant to the New York Neurological Institute to examine the infectious hypothesis. Led by Oscar Teague researchers sought to cultivate spirochetes or other organisms from the blood or spinal fluid of multiple sclerosis patients, to produce paralysis in animals by injecting them with this fluid, and to demonstrate the existence of the same spirochetes in the blood or organs of the paralyzed animals. Teague reported in 1922 that they had

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[88] Teague, "Bacteriological Investigation of Multiple Sclerosis," 122. McAlpine, *Multiple Sclerosis*, 259.

[89] Teague, "Bacteriological Investigations of Multiple Sclerosis," 122.

[90] Ibid., 122.

[91] Ibid., 123.

"practically negative results."[92] The research was continued by the College of Physicians and Surgeons at Columbia. Due to the conflicting reports of the previous nine years, in 1922 the Commission of the Association for Research into Nervous and Mental Diseases concluded that the etiological factor remained "almost completely in the dark."[93] The argued that multiple sclerosis was, "not a familial disease, and it is not inherited . . . but in the ancestry there is often evidence of neuropathic stock?"[94] Further, that:

> the work of Teague in searching for a microorganism, especially a spirochete, in cases of multiple sclerosis, has yielded a negative result. He was unable to confirm the findings of European observers in this regard. The Commission thinks that a negative result, while not as valuable as a positive one, nevertheless, in view of the histological character of the pathological process of multiple sclerosis, must induce a cautious appreciation of the finding of micro-organism in this disease.[95]

Bacteriological investigations continued through the thirties with negative results.[96] Though these negotiated statements engage in a kind of double-speak that seems to try to have it both ways, there does seem to be a subtle attempt to downplay the rather more solid epidemiological, racial, and ethnic evidence and still hope, however cautiously, that there might be a micro-organism responsible yet found.

The epidemiological evidence in terms of ethnicity and race was only confirmed from the 1910 to the 1950s. American neurologist Tracy Jackson Putnam wrote in 1947

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[92] Ibid., 124-125.

[93] Ibid., 131.

[94] ARNMD Commission, "The Conclusions of the Commission," *Multiple Sclerosis* (1922), 47.

[95] ARNMD Commission, "Conclusions of the Commission Concerning the Pathology of Multiple Sclerosis," *Multiple Sclerosis* (1922) 208.

[96] McAlpine, *Multiple Sclerosis*, 257-260.

that,

> multiple sclerosis appears to be a disease of cold, damp climates. It is commonest in northern Great Britain, the Baltic countries; and, of the United States in the northern Atlantic coast, the Great Lakes states, and the northern plains.[97]

Putting it more succinctly, Douglas McAlpine, in his textbook, *Multiple Sclerosis* (1955) in the first sentence of the book stated: "multiple, or disseminated, sclerosis ranks high among organic nervous diseases affecting the white races."[98] Because of the geographic, racial, and ethnic epidemiological data and the etiological uncertainty the same data could be interpreted in different ways depending on the social and cultural context.

For example, increasing numbers of researchers returned to the possible hereditarian etiological influences of the *Sâlpètrian* school in the mid-1940s and 1950s. This was before the discoveries of Watson and Crick would make their impact after 1954.[99] However, just as researchers returned to the hereditarian hypothesis, physicians reported an apparent epidemic of multiple sclerosis in the Faroe Islands off of Scotland during World War II and the decades that followed it. Researchers presumed that the large numbers of soldiers on the island during the war had introduced the disease.[100]

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[97] Tracy Jackson Putnam, Chairman, Medical Advisory Board, et al., *Multiple Sclerosis, Diagnosis and Treatment: Manual of Information for Use of Physicians Only, first edition* (New York: National Multiple Sclerosis Society, 1 Sept 1947), 8, in *Putnam Archives*, Box: 1, Folder: Publications #143.

[98] McAlpine, *Multiple Sclerosis*, v., 13-19.

[99] See, James D. Watson, *The Double Helix: A Personal Account of the Discovery of the Structure of DNA*, ed. Gunther S. Stent (New York: W.W. Norton and Company, 1980).

[100] McAlpine, *Multiple Sclerosis*, 21-30. W.I. McDonald, "Multiple Sclerosis," in *Cambridge World History of Human Disease* (New York: Cambridge University Press, 1993), 885.

This was the most suggestive evidence to date concerning a possible infective agent. Yet, this did not lead to clear resurgence of laboratory studies seeking to find the infectious agent as occurred in the 1910s and 1920s.

One might argue that after the bacteriological discoveries of the 1880s and 1890s it was only natural that researchers would turn to bacteriology and the doctrine of specific etiology in the 1910s; and a generation of negative results, from the 1910s to the 1930s, led orthodox researchers simply to abandon the infectious disease hypothesis in the 1940s. However this cannot explain why it took a full generation for widespread bacteriologically oriented research on multiple sclerosis to emerge only in the 1910s. Originally, multiple sclerosis like most neurological conditions of that period was considered an inherited degenerate endogenous condition. Yet at the very height of biologically reductionist thought in both the neuroscientific and popular mind from the 1880s to the 1920s, multiple sclerosis split off from other neurological conditions and cut against the grain of the larger neuroscientific paradigm. This splitting occurred because of the impact of the epidemiological knowledge that it was a malady that principally affected the "white races." The return to hereditarian influences in research in the 1940s happened in spite of the evidence from the Faroe Islands epidemic. The collapse of eugenics and the biologically reductionist social thought allowed physicians to interpret the same epidemiological evidence about multiple sclerosis in a different way.[101]

IV
WHAT DO WE DO ABOUT IT?

Obviously if the etiology remained a mystery, therapy would be a problematic

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[101] See Carl N. Degler, *In Search of Human Nature: The Decline and Revival of Darwinism in American Social Thought* (New York: Oxford University Press, 1991). George Cotkin, *Reluctant Modernism: American Thought and Culture, 1880-1900* (New York: Twayne Publishers, 1992). Rosalind Rosenberg, *Beyond Separate Spheres: Intellectual Roots of Modern Feminism* (New Haven: Yale University Press, 1982).

affair at best. Wide experimentation with different remedies is not necessarily remarkable. What is curious is that from the 1870s through the 1950s many therapies persisted for decades even though they were not proven effective nor even believed to be particularly helpful. How is it that treatments, even in the 1950s, at the height of the promise and prestige of scientific medicine could persist for as long as twenty years, without clear clinical proof of efficacy, then disappear?

Physician Meredith Clymer of the University of Pennsylvania reported in 1870 that electricity, blisters, counter-irritants, ergot, arsenic, belladonna, chloride of gold, phosphate of zinc, and nitrate of silver had all been tried "without good effect." Tonics, sulphur baths, and hydrotherapy had benefitted some. Because of the believed "diathetic" nature of the condition, Clymer recommended trying the application of a "constant galvanic current."[102] In 1871 leading American neurologist William Hammond also recommended the application of a galvanic current along with chloride of barium in doses of a grain three times a day, and *hyoscyamus* in the form of tincture from one to two drachmas three times a day. He also advised the use of cod-liver oil, iron, strychnia, passive exercise, nutritious food, two glasses of wine, and the avoidance of emotional excitement or mental labor. He listed several patients for whom the treatments had seemed to help. [103] In 1873 New York Professor of Opthamology Henry D. Noyes tried bromide of potassium with no effect.[104] Also in 1873 Minnesota physician C.H. Boardman echoed Hammond's suggestions and added the benefits of massage. However

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[102] Clymer, "Notes on the Physiology . . .," 255.

[103] Hammond, "Multiple Cerebral Sclerosis," 141,150. Hammond, "Diffused Cerebral Sclerosis," 143-144.

[104] Noyes, "A Case of Supposed Disseminated Sclerosis . . ." 44.

Boardman concluded: "nothing very satisfactory can be said concerning treatment."[105] British physician Thomas Buzzard reported in 1875: "as regards treatment . . . iodide of potassium was being given to him, not, however, with much hope that it would do good."[106] E.C. Seguin, Professor of Diseases of the Mind and Nervous System, College of Physicians and Surgeons, Columbia University, reported in 1875 that as a result of treatment with ergot and an occasional saline cathartic his multiple sclerosis patient "thought he was improving, but in fact, had not so good use of his limbs as before."[107] The treatments listed above continued to be used during the 1880s and 1890s.[108] Yet late nineteenth-century orthodox physicians acknowledged that "treatment thus far has proven entirely fruitless." Nevertheless, the same treatments continued to be used from the 1870s through the 1890s. Why?

As Charles Rosenberg as shown, early and mid-nineteenth century medical therapies, "worked" in terms of the ecology of knowledge of disease in America at that time.[109] This was true for the therapies prescribed for multiple sclerosis from the 1870s through the 1890s. Charcot thought "an impaired and irregular conduction of nerve-impulses to exist in sclerosed areas . . ."[110] As California Professor of Clinical Medicine

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[105] Boardman, "Progressive Multiple Cerebro-Spinal Sclerosis," 257.

[106] Buzzard, "A Case of Disseminated Sclerosis . . . ," 124.

[107] Seguin, "A Contribution . . . ," 283-284.

[108] See Hughes, "Inability to Gargle . . . ," 472. Butler, "Disseminated Sclerosis . . . ," 151. Dercum, "Multiple Sclerosis . . . ," 125. Hirschfelder, "Disseminated Sclerosis, 447-448.

[109] Charles E. Rosenberg, "The Therapeutic Revolution: Medicine, Meaning, and Social Change in Nineteenth-Century America," in *Explaining Epidemics and other studies in the history of medicine* (New York: Cambridge University Press, 1992), 9-31.

[110] Dercum, "Multiple Sclerosis . . . (1893)," 125.

Joseph Hirschfelder put it in a clinical lecture in 1882:

> For the sake of illustration, we may look upon the nerve fibre as a tube having a valve at the sclerotic part, and the nervous influence that passes through it a fluid. Now this nervous fluid passes down from the brain unobstructed until it reaches the imaginary valve. Its passage is prevented until sufficient fluid accumulates to overcome the valve. The fluid passes on and the valve again closes and again obstructs the onward flow of new nervous fluid, until sufficient has collected to overcome it.[111]

Thus, to prevent paralysis, the application of a constant galvanic current was thought to allow the 'nervous fluid' to flow unimpeded through the sclerotic patches. Ergot was thought to stimulate the nervous system. Indeed, in excessive doses it can induce cerebro-spinal symptoms including spasms and cramps. Silver nitrate was used as an anti-infective in case the sclerotic scars were the secondary result of some infection or toxic poisoning. Belladonna was used to temper the spastic motor symptoms. Thus, the medical therapies deployed made sense in terms of the ecology of knowledge of disease at the time. However, unlike Rosenberg's argument for the early nineteenth-century, i.e., therapies often appeared to "work" because most diseases are self-limiting, the late nineteenth-century clinicians knew that this condition was marked by remissions; so, they were not beguiled into thinking that their drugs were "curing" the patients. Yet, the treatment of multiple sclerosis in the last third of the nineteenth-century continued with same armamentarium despite the persistent knowledge for over thirty years that the therapies did not really "cure" or even help the patients much.

A possible explanation for this phenomenon emerges if we compare the therapeutic behavior of mid-twentieth-century physicians toward multiple sclerosis, at the height of the promise and prestige of scientific medicine, with the behaviors illustrated

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[111] Hirschfelder, "Disseminated Sclerosis," 447-448.

above of late-nineteenth-century clinicians.[112] The evidence for this behavior is taken from my analysis of approximately three hundred patient records that span the years 1938 to 1959. They are from the practice of a leading neurologist of his day, Tracy Jackson Putnam.

Putnam had an impressive medical pedigree. His great-grandfather was Dr. James Jackson, Professor of Physics at Harvard and a founder of Massachusetts General Hospital. His uncle, Dr. James Putnam was the first professor of neurology at Harvard. His father, Dr. Charles Pickering Putnam was a well-known Boston physician. Tracy Jackson Putnam (1894-1975) was born in Boston and received his medical degree from Harvard and graduated first in his class in 1920. Throughout the twenties he trained at Johns Hopkins, Massachusetts General, Boston City, and under Barends Brouwer at the Binnegastuss in Amsterdam. His training also included neurosurgery under Harvey Cushing at Peter Bent Bingham Hospital. He held professorships at Harvard and Columbia and was a director of the New York Neurological Institute in the 1940s. From 1940 to 1957 he was Editor in Chief of the *Archives of Neurology and Psychiatry*. He was the first director of the Medical Advisory Board of the National Multiple Sclerosis Society in the late 1940s. In 1947 he moved to Beverly Hills, California to set up private practice and become Chief of the Department of Neurosurgery at the Cedars of Lebanon Hospital, Los Angeles, California. [113] Tracy J. Putnam was an accomplished, orthodox, elite physician who personified the mid-twentieth-century American medical establishment.

Putnam was also an active researcher publishing scientific papers from the 1920s

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[112] The therapeutic advances in the 1880s and 1890s occurred principally in surgery through the introduction of antisepsis and asepsis.

[113] "Biographical Sketch, Tracy Jackson, Putnam, M.D.," ***Putnam Archives***, Box: 1, Folder: Biographical Narratives.

through the 1950s.[114] From 1931 to 1947 Putnam undertook extensive research on the possibility that vascular destruction, probably a thrombosis of venules, was a critical event that led to the destruction of the myelin sheath in multiple sclerosis and the subsequent formation of sclerotic plaques on the damaged tissue. Putnam was able to produce sclerotic plaques in dogs through an experimental production of venular thrombosis in 1936-38.[115] Putnam admitted that, "the nature of the abnormality of the blood producing the thrombosis is obscure."[116] However he set out to find an anti-coagulant that might prevent the thrombosis from occurring in multiple sclerosis patients. In 1938 he advised the use of the anti-coagulant cysteine to prevent relapses. He wrote: "I do not feel that it is an ideal anticoagulant, and we are vigorously searching for others, but I know of no other that is at all usable."[117] On similar though not identical grounds, doctors at the Mayo Clinic experimented with the vasodilator histamine in the 1940s

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[114] Putnam is probably best known as the co-discoverer, along with H. Houston Merritt, of the non-sedative anticonvulsant, diphenyl hydantoin (Dilantin; Parke-Davis), for the treatment of epilepsy in 1938.

[115] See, T.J. Putnam, J.B. Mckenna, and L.R. Morrison, "Studies in Multiple Sclerosis: The Histogenesis of Experimental Sclerotic Plaques and Their Relation to Multiple Sclerosis," *Journal of the American Medical Association* 97 (28 Nov 1931): 1591-1595. T.J. Putnam, "Studies in Multiple Sclerosis: Encephalomyelitis and Sclerotic Plaques Produced by Venular Obstruction," *Archives of Neurology and Psychiatry* 33 (May 1936): 929-940.

[116] Tracy J. Putnam, Ludwig V. Chiavacci, Hans Hoff, Hyman G. Weitzen, "Results of Treatment of Multiple Sclerosis with Dicoumarin," *Archives of Neurology and Psychiatry* 57 (Jan 1947): 3.

[117] Tracy J. Putnam, New York City, to Dr. Paul Carliner, Baltimore, Maryland, (13 May 1938), Letter, *Putnam Archives*, Box: 26, Folder: Wr,JP. See also, Tracy J. Putnam, New York City, to Dr. Paul Carliner, Baltimore, Maryland, (13 Feb 1939), Letter, *Putnam Archives*, Box: 26, Folder: Wr,JP. Tracy J. Putnam, New York City, to Dr. Paul Carliner, Baltimore, Maryland, (16 Mar 1939), Letter, *Putnam Archives*, Box: 26, Folder: Wr, JP.

though Putnam did not think it effective because its effects were too transitory.[118] In 1941 the discovery of the anti-coagulant dicoumarin (3,3 methylene-bis-[4hydroxcycoumarin]) was announced. Beginning in May 1942, Putnam began clinical trials with dicoumarin with an initial patient pool of 74 multiple sclerosis patients. However, 31 patients dropped out of the study for various reasons. Of the 43 cases remaining, 27 cases were grouped as cases characterized by the appearance of recurrent, acute, sharply limited attacks and remissions. The second group of 16 cases were characterized by a slow downward progression of the disease without well-defined exacerbations or remissions. Both groups of patients were treated for at least 6 months and not more than 47 months. The patients' prothrombin time was raised but doctors had to closely monitor it so that hemorrhaging would not occur. The results indicated that 23 of the 27 acute type cases did not have a acute relapse while the drug was administered. Nine cases out of the slow progressive group of sixteen cases showed no benefit from the treatment.[119]

This research would form the basis of Putnam's clinical treatment behavior concerning multiple sclerosis. Putnam standardized his treatment for multiple sclerosis in the mid-1940s. He advised patients to move to a warm, dry climate; avoid infections, injuries, pregnancy, and strong emotions. For those patients with the slow progressive symptoms Putnam would prescribe "ACTH or cortisone to stimulate the healing of lesions" but advise against using dicoumarin.[120] For those patients who experienced

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[118] Dr. Bayard T. Horton, Rochester, Minnesota, to Dr. L. Hawkins, Los Angeles, (14 Jan 1947), Letter, *Putnam Archives*, Box: 4, Folder: Ga,Jo.

[119] Putnam, et al., "Results of Treatment of Multiple Sclerosis with Dicoumarin," 1-13.

[120] Tracy J. Putnam to Dr. Galen S. Egbert, San Jose, CA (5 August 1954), Letter, *Putnam Archives*, Box 7, Folder: Eg, Sh.

acute relapses he advised "dicoumarolizing" them. This advice given to a patient's primary physician in May 1947 is typical of Putnam's standard behavior regarding this treatment:

> My usual plan is to take a prothrombin time, then give 150 mg of dicoumarin for three days, then repeat the test. If the prothrombin time has risen to thirty seconds or over (taking the normal as twenty seconds), the dose is reduced to 50 mg daily. This dose is continued as long as the prothrombin time (determined) every other day remains between thirty and forty, or adjusted to bring it within these limits. When the whole is well established, the prothrombin time may be determined only once every week or two.[121]

For the chronic, slow, progressive patients Putnam, as in this 1947 case, usually advised the following:

> As he is already incapacitated, and it is difficult for him to get about, and has a slowly progressive type of disease, I doubt if it would be good judgement to give him dicoumarin. He might be helped by the use of histamine - about 10% of chronic patients appear to derive benefit from it. Would you feel like administering this, or recommend someone who would? He might also be helped by a physiotherapist, at least as far as walking is concerned.[122]

Putnam claimed in the 1950s with respect to the slow progressive patients that ACTH and

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[121] Tracy J. Putnam, Beverly Hills, CA to Dr. L. Van Horn Gerdine, Los Angeles (8 May 1947), Letter, *Putnam Archives*, Box: 12, Folder: Ho, Ho. Putnam repeated this advice scores of times over the next 12 years. The examples are far too numerous to cite them all, but a few include: Tracy J. Putnam, Beverly Hills, CA to Dr. Coleman B. Hendricks, Los Angeles (31 July 1947), Letter, *Putnam Archives*, Box :23, Folder: Te, Ho. Tracy J. Putnam, Beverly Hills, CA to Dr. John B. Doyle, Los Angeles (23 Feb 1948), Letter, Putnam Archives, Box 15, Folder: Le, Do. Tracy J. Putnam, Beverly Hills, CA to Dr. Ira Cohen, New York City, (29 Mar 1948), Letter, Putnam Archives, Box 14, Folder: Le, Cl. "Patient Exam Record," (20 June 1949), Putnam Archives, Box 13, Folder: Ka, Le. Tracy J. Putnam, Beverly Hills, to Major J. W. Sumner, Medical Corps, Denver, CO (16 Aug 1950), Letter, Putnam Archives, Box 8, Folder: En, Je. Tracy J. Putnam to Dr. Espey F. Cannon (18 Jan 1951), Letter, Putnam Archives, Box 26, Folder: Wa, Ca.

[122] Tracy J. Putnam, Beverly Hills, to Dr. Samuel W. Weaver, Santa Ana, CA (12 May 1947), letter, *Putnam Archives*, Box: 14, Folder: Kn, Or.

cortisone had "produced an improvement in static symptoms in about 70% of my cases."[123] Here we begin to see what would be an endemic problem in evaluating treatments for multiple sclerosis. How could one determine the effectiveness of a treatment when spontaneous remissions were known to occur in figures that approached 70% of all cases?

For example a 1939 study of 1,407 patients found that fever therapy had helped in 60% of the cases, quinine had helped in 58%, and arsphenamine in 45%. However, in untreated cases, spontaneous remission occurred in 69% of the cases.[124] This would prove to be an enduring problem for clinicians and for the dicoumarin treatment. Writing in 1955, leading British neurologist Douglas McAlpine said:

> Ideally the object of any long-term treatment of multiple sclerosis is the prevention of relapses. Short of this, any form of treatment shown to modify both their frequency and severity to such an extent as to defer or even prevent the onset of progressive paraplegia would be a substantial therapeutic gain. However, as a result of the considerable variation in the frequency of the relapses and because of the capricious behaviour of the disease, the available data are so uncertain that it would be difficult to substantiate such a claim. For example there is no general agreement on the average relapse rater per year.[125]

Putnam's treatment was by no means hegemonic and he knew it. In August of 1950 he recommended the use of dicoumarin to an army physician and added:

> I realize of course that there are many schools of thought concerning the treatment of multiple sclerosis and that your situation at Fitzsimmons may impose limitations of which I am ignorant, and naturally I do not wish to

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[123] Tracy J. Putnam to Dr. Christian Hermann, Los Angeles, 15 Oct 1957), Letter, *Putnam Archives*, Box 13, Folder: Ka, Fr.

[124] McAlpine, *Multiple Sclerosis*, 193.

[125] Ibid., 205.

intrude into the treatment of the case.[126]

Sometimes physicians would refuse to administer dicoumarin because there was no therapeutic uniformity among neurologists with regard to the treatment of multiple sclerosis in the 1950s.[127] As McAlpine put it in 1955:

> lack of uniformity in the treatment of multiple sclerosis is due first to our ignorance of its cause, secondly to the fact that no available form of therapy can be relied upon to modify its course, and thirdly to the difficulty in assessing results in face of the natural tendency for symptoms to remit.[128]

Putnam, writing to another physician in 1953, said: "I realize that treatment is not standardized and many good neurologists would dissent."[129]

Putnam was a consulting neurologist who usually only saw patients that were referred to him by other doctors. My analysis of his patient records indicated there were three principle zones that constituted a physician to physician referral system with respect to Putnam. The first zone consisted of referrals from within the Greater Los Angeles Area running from Orange County to the south to Ventura County in the north; and from Los Angles County to Riverside and San Bernadino Counties in the east. The secondary zone included referrals from San Diego, Fresno, Bakersfield, San Jose, San Francisco, Oakland, Berkeley, Redding, and other California cities. Finally the tertiary zone included patients from Arizona, Utah, and other places in the West; included in this last

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[126] Tracy J. Putnam to Major J.W. Sumner, Denver (16 Aug 1950), Letter, *Putnam Archives*, Box 8, Folder: En, Je.

[127] MacAlpine, *Multiple Sclerosis*, 4, 190.

[128] Ibid., 190.

[129] Tracy J. Putnam to Dr. Myron Prinzmetal, Beverly Hills, (30 Sep 1953), Letter, *Putnam Archives*, Box 9, Folder: Fi, Er.

group were a few patients from Illinois, New York, and Connecticut attracted by Putnam's international reputation. There were relatively more patients in the first group, less in the second group though still substantial, and the least in the last group. Putnam often did not follow the dicoumarin treatments himself because, as a consulting physician, he sought to arrange for the patient's own physician to monitor the medication, nearly always for those who lived out of town. Putnam frequently did not follow the dicoumarin treatments even in Beverly Hills or Los Angeles.[130] For many patients, Putnam was the doctor of last hope.

Many physicians dissented from the multiple sclerosis therapies and maintained an attitude of therapeutic nihilism towards multiple sclerosis. One concerned family member wrote to Putnam in 1955 seeking advice: "we are in a predicament with <John Doe>. Should we keep him at the V.A. Hospital where the attitude towards M.S. is essentially nihilistic, or should we attempt further medical treatment with rather limited funds?"[131] The patients were very active, often going through multiple doctors till someone "did something."[132] Writing to Putnam in 1946, one patient reported that,

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[130] Tracy J. Putnam to Dr. Marcus H. Rabwin, Beverly Hills, (3 March 1948), Letter, *Putnam Archives:* Box: 4, Folder: Br, Aa. Tracy J. Putnam to Dr. George Koch, Anaheim, CA, (10 December 1947), Letter, *Putnam Archives*, Box: 2, Folder: Ar, Vi.

[131] Le.Co., Redwood City, CA, to Putnam 29 Jun 1955), Letter, Putnam Archives, Box 6, Folder: Co., Al., McAlpine, *Multiple Sclerosis*, 190. Hinton D. Jonez, as told to Miriam Zeller Gross, *My Fight to Conquer Multiple Sclerosis* (New York: Messner,1952), 160.

[132] Putnam's patient files contain large numbers of letters from patients to Putnam reporting that they had been to other doctors who would not treat them. Often they or a family member would read about Putnam in the popular press and request an appointment with him. See for example, He.Bu, Long Beach, CA to Putnam (3 Jun 1947), Letter, *Putnam Archives*, Box 4, Folder: Bu, He, Mrs. Ho. Te., Los Angeles, to Putnam (27 Feb 1948), Letter, *Putnam Archives*, Box: 23, Folder: Te, Ho. Jo.At., Long Beach, CA (22 Mar 1948), Letter, *Putnam Archives*, Box 2, Folder: At., Jo. El. Je., Santa Monica, CA, to Putnam (10 May 1948), Letter, *Putnam Archives*, Box 12, Folder: Je., El. Je.En., Denver, CO, to Putnam, (31 Jul 1950), Letter, *Putnam Archives*, Box 8, Folder: En, Br. Vi.Cr., Fresno, Ca (26 Dec 1950), Letter, *Putnam Archives*, Box 6, Folder: Cr.,

> I have been to quite a few doctors but until recently they have been uncertain as to the real cause. In the past 6 months I have been to the Sansum Clinic in Santa Barbara twice where my trouble was called 'Multiple Sclerosis'. They told me of an article in *Time Magazine* where I learned of your name. Needless to say I am very anxious to see you and get help if possible.[133]

Another patient writing to Putnam in 1948 said that,

> I have not had a doctor since 1945, when the one who examined me said that my case was incurable . . . I hesitate to ask him for a recommendation to you . . . If it is possible for me to visit you without being recommended I should be very happy to do so.[134]

This patient activism created an intense demand for therapies.

When this demand was met, the treatment participated in not only a scientific rationale but also in a moral imperative constructed through a "cluster of humanitarian narratives" that "created sympathetic passions" and "bridged the gulf between facts, compassion, and action."[135] Remember, most multiple sclerosis patients experience the first onset of symptoms between twenty and forty years of age. The individual and familial consequences would often be tragic. Many young patients attempted suicide or lapsed into deep depressions upon receiving their diagnosis. British neurologist Douglas McAlpine, writing in 1955, said:

> Formerly every effort was made to keep the patient with multiple sclerosis

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[133] St. Le., Glendale, CA, to Tracy J. Putnam (30 Dec 1946), Letter, *Putnam Archives*, Box: 14, Folder: Le, St.

[134] Vi.Bu., Glendale, CA, to Putnam (2 Jun 1948), Letter, *Putnam Archives*, Box 4, Folder: Bu., Vi.

[135] Thomas W. Laqueur, "The Humanitarian Narrative," in *The New Cultural History*, ed. Lynn Hunt (Berkeley: University of California Press, 1989), 179.

> in ignorance of the diagnosis, and it would be generally agreed that this policy should be continued in the case of the young unmarried adult . . . In answering questions as to the advisability of marriage, a combination of judgement, tact, and frankness is required . . . Whether or not patients should be told the diagnosis to allow full discussion of their problems must depend on age and moral fibre.[136]

The patient examination records of Putnam contain a telling mixture of medical and moral narratives. Not only would they include the patient's medical history and physical examination but also the human consequences of the patient's disease and letters from patients and family members. For example, in one record the usual notes concerning routine physical exams are intermixed with notes about the patient's personal struggles. The entry Putnam wrote in this 26 year-old man's examination record for 19 January 1956 was: "passed exams with c's!;" for 31 May 1956: "graduated from Notre Dame, think's he'll get in UCLA"; on 27 September 1956: "comes in, after being in Syracuse-had anxious summer because he didn't do well on law school exams. Thinks he will work in political science at UCLA"; the patient's occasional relapses and remissions are reported and the young man gradually had to abandon his college career because of his increasing physical disability and resultant dependency.[137] The 1946 patient record for one young Californian with multiple sclerosis recorded that after experiencing sudden blindness in his right eye, the patient became discouraged because of his medical condition and the financial difficulties brought on by his disease; so, he shot himself in the chest.[138]

A steady stream of letters from patients and their families flowed into Putnam's

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[136] McAlpine, *Multiple Sclerosis*, 192-193.

[137] Patient Examination Record, pages 1-10, *Putnam Archives*, Box 8, Folder: Fi., Ha.

[138] Patient Exam Record (3 Dec 1947), *Putnam Archives*, Box 2, Folder: Ar, Vi.

office. One Canadian wrote to Putnam concerned because his brother, afflicted with multiple sclerosis, was deeply depressed.

> More recently he has not been himself. He is very morbid and moody, and is constantly worrying about his sickness. There is no doubt he is very much upset, and life appears to have become meaningless to him.[139]

Putnam consistently responded directly to the family and patient petitions with sympathy and understanding. So, when a young person suddenly struck down in the prime of her life, desperately seeking any possible treatment, almost literally "banged down the door" for help, what else could a compassionate humanitarian do but prescribe a treatment, even if the possibility of amelioration was remote? For example, even though Putnam had little faith in histamine treatments, writing to hospital administrator Arthur C. Bockstahler of North Hollywood on 17 May 1954, he counselled:

> I recently talked to Mrs. <Jane Doe> whom you have been treating for us for multiple sclerosis. She has heard of improvement from histamine given by iontophoresis and I should be happy to have her try this although the proportion of success is low in my experience. She tells me that you have been accustomed to administering it and I would be greatly obliged if you would do so according to your usual technique in her case.[140]

That Putnam was not alone in his behavior is indicated by a November 1952 article in *Newsweek* which reported that,

> Because of the mystery surrounding the cause of multiple sclerosis, physicians are likely to shrug off their patients with a 'sorry, nothing can be done' attitude. The neurologists who discussed Dr. Brickner's paper at the Academy meeting unanimously applauded his professional courage for scorning this 'therapeutic nihilism,' and acting experimentally to assure MS victims that there may be some relief, even temporary, for their tragic

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[139] Hi., Co., Winnipeg, Canada, to Putnam (13 Feb 1947), Letter, *Putnam Archives*, Box 5, Folder: Co., No.

[140] Tracy J. Putnam to Arthur C. Bockstahler, North Hollywood, CA (17 May 1954), Letter, *Putnam Archives*, Box 5, Folder: We., Gr.

crippling disease.

Dr. L.M. Eaton writing to Dr. Louis W. Nie of Indianapolis, Indiana on 4 May 1951, concerning a patient reported that,

> I found the patient to be justifiably disturbed about the prognosis, and I tried to be as reassuring as possible regarding the occurrence of a remission. It seemed to me that the patient was in need of rather active treatment particularly from the standpoint of the psychological support it would give him.[141]

Thus, the physicians who would treat multiple sclerosis did so partly because of a moral imperative to "do something." Of course these treatments were rooted in the mid-century biomedical science; but in terms of the structure of their scientific and moral logic, and in their verifiableness, the patterns of therapeutic behavior towards multiple sclerosis at the height of the prestige and promise of scientific medicine in 1955 seem quite similar to those in 1875. Putnam was often the last chance of desperate patients who had consulted with a long series of physicians who expressed a therapeutic nihilism toward multiple sclerosis.

Another physician of last chance in the early 1950s was Dr. Hinton D. Jonez who treated nearly two thousand multiple sclerosis patients at St. Joseph Hospital in Tacoma, Washington between 1947 and 1952. Jonez believed the destruction of the myelin sheath was initiated by an allergic reaction so he prescribed histamine intravenously hoping that its vasodilation effect would prevent damage to the myelin sheath.[142] Clinical trials with

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[141] L.M. Eaton, M.D., to Louis W. Nie, M.D. (4 May 1951), Letter, ***Putnam Archives***, Box 8, Folder: Fi., Er.

[142] Hinton D. Jonez, as told to Miriam Zeller Gross, ***My Fight to Conquer Multiple Sclerosis*** (New York: Julian Messner, Inc. 1952), 7-40.

histamine were conducted at the Mayo Clinic from 1942 to 1944.[143] By the later 1940s most mainstream neurologists abandoned histamine as a useful treatment. As Harry H. Larson wrote in 1952:

> Histamine treatment, while in many instances causing improvement during intravenous administration, has no sustained effect. Relapses occur in the majority of cases within a short time. Although prolonged administration seems to produce no serious ill effects, the inconvenience and difficulty of the procedure make it unfeasible over long periods of time.[144]

One 43 year old California woman who was seeing a number of physicians for her condition told one doctor that, "she was going to Tacoma, Wash., for the 'histamine treatment.' She stated that the assumption is that the etiology is 'allergic.'"[145] This jumping from physician to physician by patients was common as they sought any therapy that might have a chance of success. Putnam, writing to the daughter of a California patient who was planning to go to Tacoma wrote in 1950:

> I am sorry to hear that your mother is not so well. The treatment which is being tried at Tacoma is similar to that used in many other clinics and could certainly be given at Vanderbilt. Most of us have had only indifferent success with it, but it is certainly harmless.[146]

Though most neurologists had abandoned histamine treatments by the late 1940s Jonez

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[143] Bayard T. Horton, Rochester, Minnesota, to Dr. Hawkins, Los Angeles, California, 14 January 1947. *Putnam Archives*, Box 4, Folder Ga, Jo.

[144] Harry H. Larson, "Incidence of Symptomatology and Recent Therapy in Multiple Sclerosis" (M.D. thesis, University of Wisconsin, 1952), 17.

[145] Sam Peck to Andrew F. LoPinto, San Diego, California, (24 September 1951), Letter, *Putnam Archives*, Box 11, Folder Ha, Ma.

[146] Tracy J. Putnam, Beverly Hills, to Mrs. V.L. Al. North Hollywood, (6 December 1950), Letter, *Putnam Archives*, Box 15, Folder Ll, Lo.

was able to draw patients from around the country because of the vigorous proactive behavior of patients and the decentralized structure of the American medical profession. Jonez was a general practitioner and a self described simple country doctor. He had wandered into multiple sclerosis treatment because of an interest in allergies and because of his personal confrontation with the tragedy of multiple sclerosis. He adopted a treatment that most mainstream neurologists had abandoned by the mid-1940s and because he was professionally and geographically isolated he could begin to give the treatment when most had already abandoned it. The decentralized disciplinary structure of the American medical profession meant that this ineffective therapy could continue to be prescribed well into the 1950s. The willingness of patients to try different doctors and therapies around the country combined with the decentralized structure of the American medical profession assured that there would be a proliferation of unproven therapies that would be difficult to evaluate. This was **not** driven by economic motive. I found many instances in which doctors would refuse to give any treatment because they believed none would work and it would have been unethical to use them. The question is: why was Jonez considered a quack by the neurological community and the National Multiple Sclerosis Society while Putnam was not, considering that neither histamine nor dicoumarin had really been proven to be effective? Putnam's impressive medical pedigree and the fact that he was the co-discover of Dilantin (a medication to prevent seizures in epilepsy) surely helped to prevent any invidious comparisons.

Those physicians who would attempt to treat multiple sclerosis were decidedly in the minority in 1946. Indeed the dominant response of the medical profession was perceived by the patients to be one of therapeutic nihilism. Often, patients in mid-twentieth century United States would not accept this answer. Indeed this attitude on the part of many physicians fractured the emotional relationship between physician and patient and caused patient deference to decline. Patients like many less conservative

neurologists constructed a humanitarian narrative that something must be done. Because it was a medical mystery, patients were able to devise a political, social, and cultural remedy. In other words, if the medical community by-and-large would not make multiple sclerosis a priority the patients themselves would organize to do something about it. This movement shows how deep the cultural change, which had occurred by 1946 in the United States, affected the assumptions and behavior of lay health activists and how at the height of the prestige and promise of medical science at mid-century the beginning of the decline of patient deference had begun. The half-century of dramatic success in the medical sciences combined with larger cultural changes meant that, when no medical solution was forthcoming, patients began to demand one like they might for any other public works project.

Patients and their families when confronted with the mystery of the cause and cure of multiple sclerosis in the postwar United States refused to accept the medical nihilism toward the condition. In an American cultural model rooted in the reform movements of the early twentieth-century they collectively organized into a private voluntary organization with the goals of mobilizing the medical community, raising funds for research, and raising public consciousness about the condition. For those with multiple sclerosis, 1946 marked a critical turning point in their personal experience of the disease. Before this time multiple sclerotics were usually isolated, alone, and hopeless. After this year multiple sclerosis became a concept around which a rapidly growing lay community would form a collective identity, and become a political force within the biomedical research world. Though multiple sclerosis had no cure by the end of the 1950s the collective experience of multiple sclerosis patients changed dramatically because of salutary effect of working together and feeling useful again in a crusade for health and because the victims drew courage, hope, and strength from one another.

Multiple sclerotics tended to be hidden from one another before 1946 because it

was not a reportable disease. Hospitals were reluctant to, and often, would not admit MS patients because they believed there was nothing they could do for the patients.[147] As Putnam put it in 1949,

> The mental and emotional state of those who know that they have the disease is pitiful, since they are aware that they have become or are bound to become a burden on their family, in many cases unsupportable, and that they are bound to be progressively incapacitated and ultimately bedridden, if not actually, killed, by the disease . . . In consequence of this situation, doctors are loath to inform the victim or his family of the fact that he has the disease. He is frequently told that he has something else.[148]

The doctor of the afflicted brother of Sylvia Lawry, the founder of the National Multiple Sclerosis Society, would often refuse to see him when new symptoms would arise. This attitude was indicative of the general therapeutic nihilism among physicians. Often doctors would refuse to give a diagnosis because of the emotional cost and because it was feared that patients might lose their jobs or be marginalized in other ways. This helped to keep the disease hidden as the earlier stigmatizing associations had. And when a diagnosis would be made, patients would be isolated from one another because of the general lack of consciousness about how widespread the condition really was.

The level of frustration of patients and their families is illustrated by the following statements from a 1949 congressional hearing on multiple sclerosis where the ambivalent and fractured relationship between physicians and patients can be seen. Senator Charles W. Tobey of New Hampshire sponsored the legislation which would have set up an institute for the study of multiple sclerosis. In his opening statement Senator Tobey, whose daughter had multiple sclerosis said,

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147 Congress, House of Representatives, Subcommittee of the Committee on Interstate and Foreign Commerce, *National Health Plan*, 22 June 1949, 618.

148 Ibid., 619.

> we might as well be candid about it, practically nothing of medical value is presently known about the cause, control, or effective treatment of multiple sclerosis. For all intents and purposes our knowledge concerning the disease is practically the same it was 80 years ago.

Mrs. Lou Gehrig testified that,

> it is a tragic fact that my testimony on this subject is almost as acceptable as that of any doctor in the land. This is not an indication of my erudition. It is an indication of how little is known concerning multiple sclerosis--even by the doctors who are most interested in it. [149]

Ralph I. Straus, President of the National Multiple Sclerosis Society said that,

> in the 80 years that have elapsed since the eminent French neurologist, Jean Martin Charcot, first identified the disease now known as multiple sclerosis, little, if any important progress has been made in the field concerning its cause and effective treatment.

Since the patients perceived that the biomedical community was not doing enough to find a cure for the disease, the patients had taken matters in their own hands. Physicians had a response to this conflict with patients and among themselves. Physicians would deploy a narrative myth of progress about multiple sclerosis to contain social conflict. For example, later in the same hearing Tracy Jackson Putnam responded to the battering the lay activists were dealing out to the medical community. Putnam retorted that, "it is not quite fair to say that we have learned nothing. Actually, a great deal of information has been accumulated, and it is now becoming clearer and clearer the directions in which studies should be pursued." Putnam then recited tale of progress from Carswell and Cruveilhier, through the Germans, to Charcot. He went on to argue that "much information gradually accumulated during the remainder of the nineteenth-century . . ." As I discussed earlier there was no linear research connection between Cruvelhier, Carswell, and Charcot. The work of the Germans did not advance clinical understanding

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[149] Ibid., 11.

and they did not construct a new disease entity. So, in a real sense they were not talking about the same phenomenon that Charcot was elucidating in the 1880s. And, even in the 1920s neurologists could not agree if multiple sclerosis was really a singular disease. By the 1950s questions of etiology and treatment were almost completely in the dark still. This narrative myth of progress is deployed at the beginning of scientific texts about multiple sclerosis and serves to contain the conflict between different researchers and between different research communities. It also serves to mystify the lack of scientific progress about this most mysterious disease. This may have served a vitally necessary integrative function for the research community concerned with this problem. All cultures need foundational myths to order their chaotic universes. The invocation of the myth as ritual in clinical lectures and texts served to creatively imagine a transnational medical community when actually the work of the science was done in much smaller competitive national communities. The myth's function was to contain conflict, intensified because of the continuing mystery of multiple sclerosis, between the disparate and contentious medical groups that inhabited this shared professional space and to contain the potential disruption of professional unity due to their often contradictory medical ideas and behaviors.[150] While this may have worked to contain conflict between physicians the patients did not accept the story as sufficient reason for hope. Instead it contradicted the promise, prestige, and actual accomplishments of biomedicine in 1950. Multiple sclerosis was the negative poster child that betrayed a larger medical impotence beyond surgical and anti-biotic intervention in 1950. Perhaps the cultural decline of

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[150] For the early years of the AIDS epidemic, Russell C. Maulitz and Jacalyn Duffin, following Mirko D. Grmeck, argue that, "as national borders were crossed--by the virus, by the theories about, by the people studying it-- the 'official chronology' explicitly became something like an out-of-court settlement. It was drafted treaty like, in appropriate elliptical phrases to mask the painful, the unresolvable, the untranslatable," in "Translators' Preface," in Mirko D. Grmeck, *History of AIDS: Emergence and Origin of a Modern Pandemic*, trans. Russell C. Maulitz and Jacalyn Duffin (Princeton: Princeton University Press, 1990), viii.

deference to physicians was inevitable considering the unrealistic promise biomedicine seemed to offer in the first half of the twentieth-century. It was probably impossible to live up to such high expectations. The seeds for the decline of deference that would break out in full force in the 1960s had taken deep roots in the 1950s.

To combat the therapeutic nihilism, the belief that multiple sclerosis was a rare disease and the hopeless, isolated, and depressing situation of multiple sclerosis victims, Sylvia Lawry had placed an ad in the New York Times in 1946 asking if anyone knew of a treatment or cure for multiple sclerosis. Hundreds of letters flowed in from around the country and soon Lawry organized meetings of patients and their families in New York City. It is most unlikely that there was a sudden upsurge in actual cases of multiple sclerosis in the 1940s. What happened was that the decades-long process of uncovering along with countervailing cultural and social forces that kept the disease off the social radar screen had built up to a point of critical mass. When Lawry began her campaign it was if a match had been thrown on dry kindling. The patient movement was ready to explode because the social forces that had served to keep the disease off the social radar screen could not keep the disease hidden any longer. The tuberculosis and polio campaigns of the early twentieth-century, the mobilizing of an institute for cancer research in the NIH in the 1930s, and the religious and ethnic sponsorship of hospitals provided a cultural model for this wave of patient activism.

The MS Society began by attempting to find and bring in as many patients and their families as possible into a private voluntary organization to fight multiple sclerosis.[151] Lawry took as her model the national polio association. She approached the polio people and asked them to take up the multiple sclerosis cause. However they were

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[151] Congress, Senate, Subcommittee on Health of the Committee on Labor and Public Welfare of the United States Senate, *National Multiple Sclerosis Act* , 81st Cong., 1st sess., 10 May 1949, 22.

unable to help because their funds were committed to polio. They did offer advice towards starting an organization: do not let the doctors be in charge because they do not know how to raise funds. By 1949, the M.S. Society had around 15,000 members nationwide. The Multiple Sclerosis Society became the center of information about the disease a social site of comfort and support. Doctors, patients, government agencies, and health care facilities would write from around the world seeking advice on multiple sclerosis. Patients lives were transformed as they became participants in the crusade to find a cure and as they drew strength from the fellowship of struggle and work with their fellow MS sufferers. The society began with the goal of patient support and it then planned a public campaign directed towards the government, the medical community, and the larger culture, to the end of curing multiple sclerosis. The MS activists combined the model of American voluntarism with the culture of the New Deal and the precedent of government involvement in science during WW2.

The MS Society lobbied the federal government to start a Multiple Sclerosis Research Institute like the institute devoted to cancer research. On 10 May 1949 a hearing was held before the Subcommittee on Health of the Committee on Labor and Public Welfare of the United States Senate to consider S. 102, the *National Multiple Sclerosis Act.* Senator Tobey argued that,

> as you look upon these people this morning and see first-hand the ravages of this disease, which brings home the fact that here in the United States in the twentieth century there is no known cause and no known cure for this disease, that the indictment and the responsibility is plain? I believe there is money enough in this Nation to equip researchers as fully and as completely as manpower will allow to get to the bottom of these diseases that are breaking the morale of men and women and destroying home life."[152]

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152 Congress, Senate, Subcommittee on Health of the Committee on Labor and Public Welfare of the United States Senate, *National Multiple Sclerosis Act* , 81st Cong., 1st sess., 10 May 1949, 15.

Tobey later added:

> So, I as a father of a sufferer, introduce this legislation. There isn't a medical man in this room, and I will include the Senator, who would not lay down his life at this moment if he could find a cure for the thing. But we are challenged. We cannot take this thing lying down. There is money enough in this country to take care of this job. When we spend $5,000,000,000 for the Marshall plan--which I voted for and when we spent $12,000,000,000 every 30 days in World War 2 to kill men and destroy capital property forever, we cannot for a moment sit back idly and say, 'We cannot appropriate whatever millions are necessary to find the cause, and the research to look into this hellish disease and to give men courage and faith to restore these things.' We may not be successful, but God will hold us responsible unless we try to do something for them.[153]

Ralph I. Straus, President of the National Multiple Sclerosis Society maintained that,

> It seems to me that consideration by a committee of the United States Senate of legislation to combat multiple sclerosis is an important social achievement. It indicates a general acceptance of the fact that disease is everybody's business, and that which is everybody's business is the business of government.[154]

Dr. C.H. Traeger, Medical Director, National Multiple Sclerosis Society argued that

> Now, as to the need for funds, they are first of all needed for basic research. Basic research means picking apart the little building blocks which make up the human organism. The men who are qualified to do that research are available; talent, genius, and interest are here. What we need is money to pay these people to do the job. If you get enough people and give them enough money you will get an atomic bomb. If you get enough people who are interested and have genius and give them the wherewithal you will get the answer. We cannot get it by ourselves.[155]

One Illinois citizen echoed the doctors call in a letter to Senator Tobey:

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[153] Ibid., 8-9.

[154] Ibid., 19.

[155] Ibid., 39.

> It is my understanding there is a bill before Congress to furnish more aid to such institutions (Kaiser Kabat Foundation in Vallejo). I am a die-hard Republican and am against all forms of Government subsidy, but when I know that only 10 percent can afford this treatment and that 90 percent are dying a slow and sure death then I shall be happy to revamp my opinion.[156]

The National Institute of Health opposed the formation of a separate institute for multiple sclerosis because of the administrative burden of a separate institute for one particular disease. As a result the MS Society changed its tactics and lobbied for the founding of a neurological institute with funding for multiple sclerosis research.[157] The lobbying of the federal government by the National Multiple Sclerosis Society resulted in several epidemiological studies of the disease by the National Institute of Mental Health from 1947 to 1951.[158] During this period a total of $241,512.23 was allocated for research into multiple sclerosis. Of this the United States Public Health Service granted $ 12, 654 for a project at Cornell University. The National Multiple Sclerosis Society provided $194,358 with $34,500 coming from other private sources.[159] Though the initial outlays were meager because of persistent lobbying by the MS Society the federal government invested more money throughout the 1950s. By 1992 the National Multiple Sclerosis Society had 400,000 members and 141 local chapters. They raised a sum of $150 million for research in 1992 alone. Only the federal government allocated more and

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[156] Ibid., 35.

[157] Congress, *National Health Plan*, 611-615. Interview, Sylvia Lawry, 10 November 1994.

[158] Congress, House of Representatives, Subcommittee of the Committee on Veterans' Affairs, *Three-Year Presumption of Service Connection for Multiple Sclerosis*, 82nd Cong., 1st sess., 20 March 1951, 130-133.

[159] Congress, House of Representatives, Subcommittee of the Committee on Interstate and Foreign Commerce, *National Health Plan*, 22 June 1949,

these federal dollars were the result of persistent lay activism and lobbying.[160]

Such extensive research in postwar America on this disease was made possible because of the collective organizing of patients, their families, and their advocates. The National Multiple Sclerosis Society was rooted in a long tradition of American voluntarism. Because patients and their advocates would not accept the nihilistic attitude of the medical community towards multiple sclerosis they formed a highly effective interest group to push their cause. One of the ways they garnered support was through the construction of humanitarian narratives of suffering and tragedy which demanded a societal response. This narrative of necessity merged with the New Deal ethos in the late 1940s. After the national experience of the New Deal in the 1930s, the federal war mobilization in World War 2, the success of the Manhattan Project, and the government's successful efforts in making penicillin during the war, lay activists expected that the federal government had a necessary and legitimate role in fostering laboratory research to fight this disease.[161]

The Society also began a public education campaign in 1947 which attempted to raise the public consciousness about the disease, to bring multiple sclerosis fully out into the open and instill hope in its victims. Sylvia Lawry enlisted the support of Madison Avenue advertising executives and the editor of Newsweek, Raymond Moley, to plan a national campaign publicize the multiple sclerosis crusade.[162]

In the late 1940s articles began to appear in *Time, Newsweek, Saturday Evening*

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[160] National Multiple Sclerosis Society 1992 Annual Report, ***Making a Difference in People's Lives***, 1-4.

[161] See Peter Neushul, "Science, Government, and the Mass Production of Penicillin," ***Journal of the History of Medicine and Allied Sciences*** 48 (1993): 371-395.

[162] Interview, Lawry, May 1994. See also, Raymond Moley, "Weapons Against a Pitiless Enemy," ***Newsweek*** (3 May 1954): 100.

Post, Cosmopolitan, Readers' Digest, Coronet, American Mercury, and Today's *Health.* In "Sentence Commuted," (1950) J. Joseph reported that for patients, "the dread diagnosis of multiple sclerosis amounted to a life-time sentence of physical incapacity--often to eventual helplessness." However, the new struggle being waged against the disease by medical institutions meant that the sentence had "been commuted to at least partial recovery . . ." Patients could expect help to "refit themselves for something near normal lives." Patient Sally Biggs was pictured at a weight machine where she did 200 sit-ups a day with weighted pullies.[163] In an article from *Newsweek* (1952) the vasodilation therapy of Drs. Richard M. Brickner and C.R. Franklin of New York is praised and the reader is instructed:

> Because of the mystery surrounding the cause of multiple sclerosis, physicians are likely to shrug off their patients with a "sorry, nothing can be done" attitude. The neurologists who discussed Dr. Brickner's paper at the Academy meeting unanimously applauded his professional courage for scorning this "therapeutic nihilism," and acting experimentally to assure MS victims that there may be some relief, even temporary, for their tragic crippling disease.[164]

26-year-old MS patient Sally Mulligan told her story in "Victory in a Wheel Chair," (1954). People often thought that Mulligan was drunk on the street early in her condition. Doctors correctly diagnosed her with MS after she went to New York Bellevue Hospital's multiple sclerosis clinic, maintained by the National Multiple Sclerosis Society. After nine months of rehabilitation and therapy she was able to return to relative normality despite her condition. She is pictured walking on crutches down a New York street, chatting with neighborhood children, and working as a receptionist in her wheelchair at Bellevue Hospital. She is shown laughing with her coworkers and is

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[163] J. Joseph, "Sentence Commuted," *Today's Health* 28, no. 5 (May 1950): 16-17.

[164] "Mysterious MS," *Newsweek* 40 (24 Nov 1952): 57-58.

quoted: "lunch hour is always fun with the girls at the hospital. Picnic style and lots of gossip about dresses and boys." She is pictured throwing her hat in the air at a party and reports that, "my boyfriend and I date once a week." She is also shown cooking and praying.[165]

The tension between the goals of mobilizing society through the image of the tragedy of the young stricken with a debilitating disease and the goal of instilling hope in victims appears in the May 1954 article in the *Saturday Evening Post.* mentioned earlier in part II. The patient Robert Grant, Jr. describes his condition and therapeutic rehabilitation with military metaphors. His therapeutic successes are flushes of victory. MS is a phantom sniper and "mystery is his iron curtain." This disease is associated with Stalin, "the Nazis and the Nips." Like victory in World War II, Grant is hopeful for victory against MS. He wrote: "for deep down inside me I know that, in this age of atomic energy, antibiotics and radioactive isotopes, a cure for my trouble is around the scientific corner." Grant is shown playing the piano and joking with nurses. All these hopeful images contrast with the pictured images of the young soldier being fed by a nurse, his confinement to a wheelchair, and his use of only two fingers. This article is typical of the tensions within this advertising campaign. On the one hand the articles sought to create a humanitarian narrative that something must be done to prevent this tragedy. On the other hand the educational campaign sought to instill hope in MS patients who were often isolated, depressed, and desperate. The articles show the tempered frustration many MS patients felt toward the biomedical research community. They thought that their disease was not receiving the attention it deserved but they did not want to alienate the biomedical community on whom they were dependent for a cure. This ambivalent relationship between patients and physicians was reflected in the

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[165] R. Moley, "Victory in a Wheelchair," *Look* 18, no. 10 (18 May 1954): 31-35.

structure of the MS Society. Lay activists were in charge of the organization and its funds. However they funnelled money through a medical advisory board not unlike the foundations in the 1920s.[166] The difference was that in the 1950s patients were becoming the patrons.

Ultimately the public relations campaign begun in 1947 and continued throughout the 1950s was successful. The MS Society persuaded the nation that multiple sclerosis was not rare but affected hundreds of thousands of people. They garnered private and federal support which made the mobilization of larger numbers of researchers possible. They also ended the isolation of MS patients who drew courage, strength, and support for their collective crusade for a cure.

IV
CONCLUSION

The way that multiple sclerosis as a concept mediated the relationship between physicians and patients changed dramatically from 1940s to the 1950s. Since the late nineteenth-century a diagnosis of MS was considered grim with no known cause or cure. Because of its polymorphous nature, what we now call MS was hidden in other nosological categories. Physicians often tried to hide the diagnosis in order to protect the patients emotionally and in terms of the social cost. Since the disease was easily stigmatizing through its association with hysteria, syphilis, and tainted heredity the true extent of the disease in the population remained hidden. Multiple sclerotics found the promise of biomedical science, at its height and prestige in the mid-twentieth century, to be unfulfilled with regard to their condition. Because of the therapeutic nihilism and social marginalization that many patients confronted, they collectively organized to

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[166] See Robert E. Kohler, *Partners in Science: Foundations and Natural Scientists 1900-1945* (Chicago: University of Chicago Press, 1991).

demand that their needs be addressed by the federal government and the biomedical research community. Lay power over the MS Society in a democratic partnership with scientists and physicians was made possible because of the way the medical mystery forced patients to question the deferential relationships of patient and physician. In the process lay activists reconfigured the cultural model of how a person with a chronic illness ought to cope with her condition by creating a communal identity of collective organizing and mutual support. The cultural construction of multiple sclerosis changed not because of any dramatic breakthroughs in terms of understanding the etiology or the cure of the disease but because of a concerted lay campaign in the 1940s and 1950s.

BIBLIOGRAPHICAL ESSAY

The history of multiple sclerosis has not received sustained treatment by professional medical historians. Only a few brief historical notes appear in medical journals and texts written by practicing neuroscientists.[167] To uncover the story of multiple sclerosis I examined articles in French, British, and American medical journals from the 1860s-1940s, including the original 1868 articles of Charcot, and medical textbooks on multiple sclerosis from the 1920s to the 1950s. I analyzed approximately three hundred patient records that date from 1938 to the late 1950s located in the *Tracy Jackson Putnam, M.D. Archives* at the University of California, Los Angeles. These records include: patient examination records, prescriptions, extensive correspondence between physicians, correspondence between patients and Putnam, financial records, and newspaper and magazine clippings. In order to protect patient confidentiality, I have cited the patient records by using the first two letters of the patient's last name, followed by the first two letters of the patient's first name. This should make it easy for anyone to find the records in the archives. I twice interviewed Ms. Sylvia Lawry, the founder of the National Multiple Sclerosis Society which was established in 1946. Finally, I read articles from the American popular press from the 1930s to the 1950s on multiple sclerosis

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[167] See W.I. McDonald, "The dynamics of multiple sclerosis: the Charcot lecture," *Journal of Neurology* 240 (1993): 28-36. W.I. McDonald, "Multiple Sclerosis," in *Cambridge World History of Human Disease* (New York: Cambridge University Press, 1993), 883-887. Alastair Compston, "Reviewing Multiple Sclerosis," *Postgraduate Medical Journal* 68 (1992): 507-516. G.E. Berrios and J.I. Quemada, "Andre G. Ombredane and the Psychiatry of Multiple Sclerosis," *Comprehensive Psychiatry* 31 (1990): 438-46. E.H. Jellinek, "Heine's illness: the case for multiple sclerosis," *Journal of the Royal Society of Medicine* 83 (Aug 1990): 516-519. Sten Fredrikson and Slavenka Kam-Hansen, "The 150-Year Anniversary of Multiple Sclerosis: Does Its Early History Give an Etiological Clue?," *Perspectives in Biology and Medicine* 32, no. 2 (Winter 1989): 237-243.

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APPENDIX

TABLE I

FREQUENCY OF MULTIPLE SCLEROSIS

IN THE UNITED STATES

YEAR	ESTIMATE	USA POPULATION
(1922).....	~ 10,000 cases	105,710,620 (1920)
	....10 per 100,000	
(1950).....	250,000 - 300,000 cases	150,697,361 (1950)
	....166 per 100,000	

Sources:
Association for Research in Nervous and Mental Diseases, ***Multiple Sclerosis*** (New York: Paul B. Hoeber, 1922), 8-12,28. Charles B. Davenport estimated 10 per 100,000 and based on the 1950 figures a rate of 166 per 100,000 is supposed. U.S. Congress, Senate, Subcommittee on Health of the Committee on Labor and Public Welfare. *National Multiple Sclerosis Act*, 81 st Congress, 1st Session, 10 May 1949. U.S. Congress. House of Representatives, Subcommittee of the Committee on Veterans' Affairs. *Three-Year Presumption of Service Connection for Multiple Sclerosis.* 82nd Congress., 1st Session, 20 March 1951. Robert Grant, Jr. "I've Got the Most Mysterious Disease," *Saturday Evening Post* 226 (22 May 1954): 26.

TABLE II

RELATIVE FREQUENCY OF PRESENTING SYMPTOMS IN MULTIPLE SCLEROSIS

SYMPTOMS	793 pts. 1949	389 pts. 1950	666 pts. 1954
Motor weakness	32%	51%	48%
Paraesthesia	11%	9%	30%
Retrobulbar neuritis	20%	14%	27%
Double Vision	13%	13%	12%
Vertigo/Vomiting	11%	4%	5%
Micturition disorder	5%	3%	5%

Source: Douglas Macalpine, *Multiple Sclerosis*, (Edinburgh: E. & S. Livingstone, Ltd., 1955), 66.

TABLE III

GERMAN AUTHORS WRITING ON MS, 1860-1870S

George Eduard **Rindfleisch**:

1856-1860 Heidelberg, Halle, Berlin . ..medical education under Virchow. He went to
1861: Heidenhaim , Breslau ..... qualified as a university lecturer in pathological anatomy.
1862: Zurich ..... lecturer in pathological anatomy in and promoted to assistant ordinarius professor in 1864.
1865: Bonn ..... ordinarius professor of pathological anatomy.

Friedrich Albert von **Zenker**:

1843-47: Leipzig ..... student
1848-49: Heidelberg
1849-1851: Leipzieg
1850: Vienna ..... pathological anatomy studies
1851: Leipzig ..... graduated with a specialty in pathological anatomy.
1851-1862: Dresden ..... Prosector at the state hospital in from 1853-1855, lecturer, thenas professor of general pathology and pathological anatomy in the surgical-medical Academy till 1862.
1862-1890s: Erlangen ..... ordinarius professor

Carl F. **Frommann**

185?-1854: Jena, Göttingen, Prague, and Vienna medical education
1856-1858: Jena ..... assistant physician at the medical clinic
1858-1860: London ..... house physician at the German hospital
1861-1870: Weimar ..... general practitioner
1870-1872: Heidelberg ..... privatdocent
1873-1874: Jena ..... privatdocent
1875 Jena ..... professorship

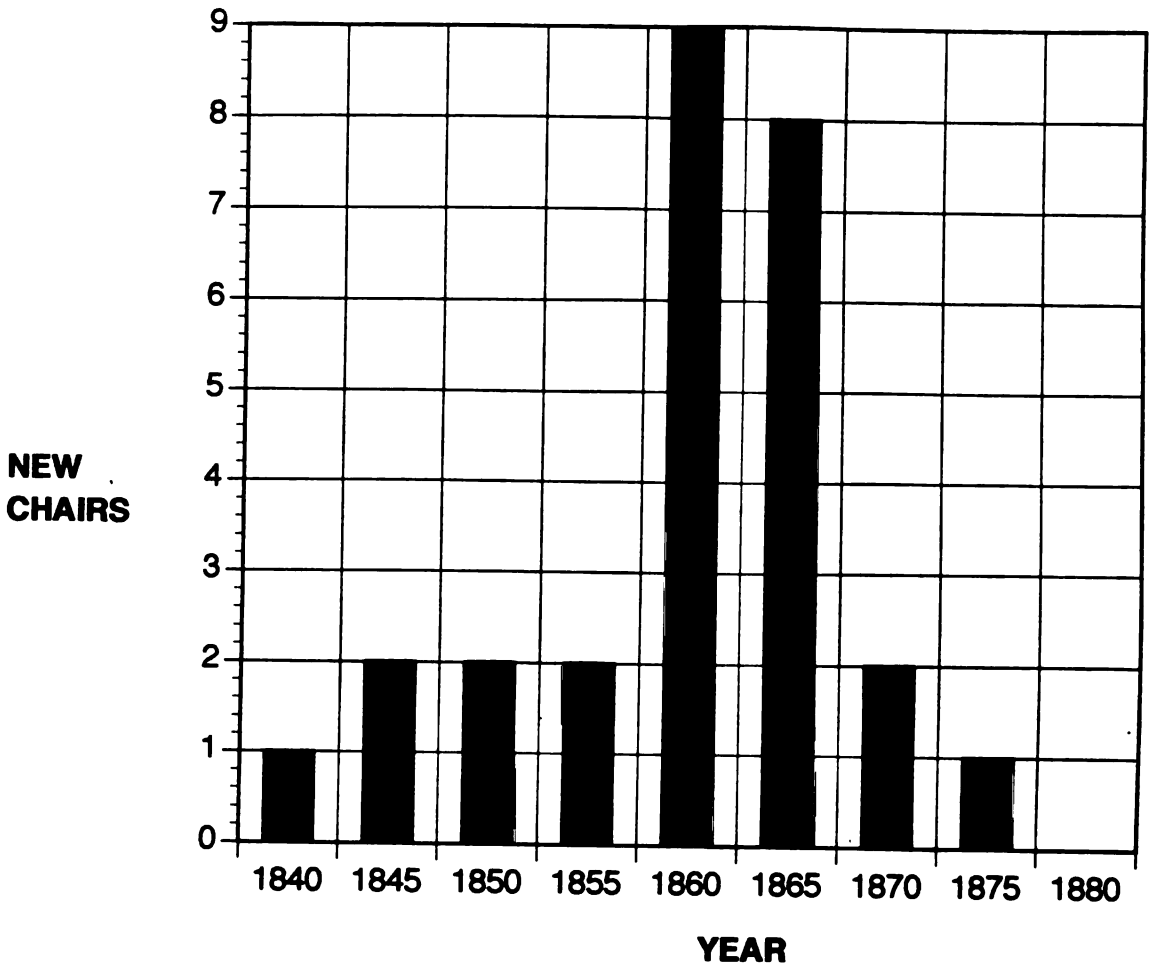
George Theodor **Valentiner**:

1843 Kiel ..... medical degree with the dissertation "Questiones duae detypho."
1849- Schleswig-Holstein ..... Provisor (Dispenser) then Head Physician at navy hospital.
1851- Kiel ..... Privatdocent.
Later **Pyrmont** ..... general practitioner in till his death 1877

Source:*Biographisches Lexikon der hervorragenden Ärzte aller Zeiten und Völker*, ed. E. Gurlt and A. Wernich (Berlin: Urban & Schwarzenberg, 1934), 633,634,693,613,614,817, 818,1034,1035.

TABLE IV

FREQUENCY OF ESTABLISHMENT OF ORDINARIUS CHAIRS IN PATHOLOGICAL ANATOMY IN GERMANY, 1840-1880



SOURCE: Robert E. Kohler, *From Medical Chemistry to Biochemistry: The Making of a Biomedical Discipline* (Cambridge University Press, 1982), 12.

TABLE V

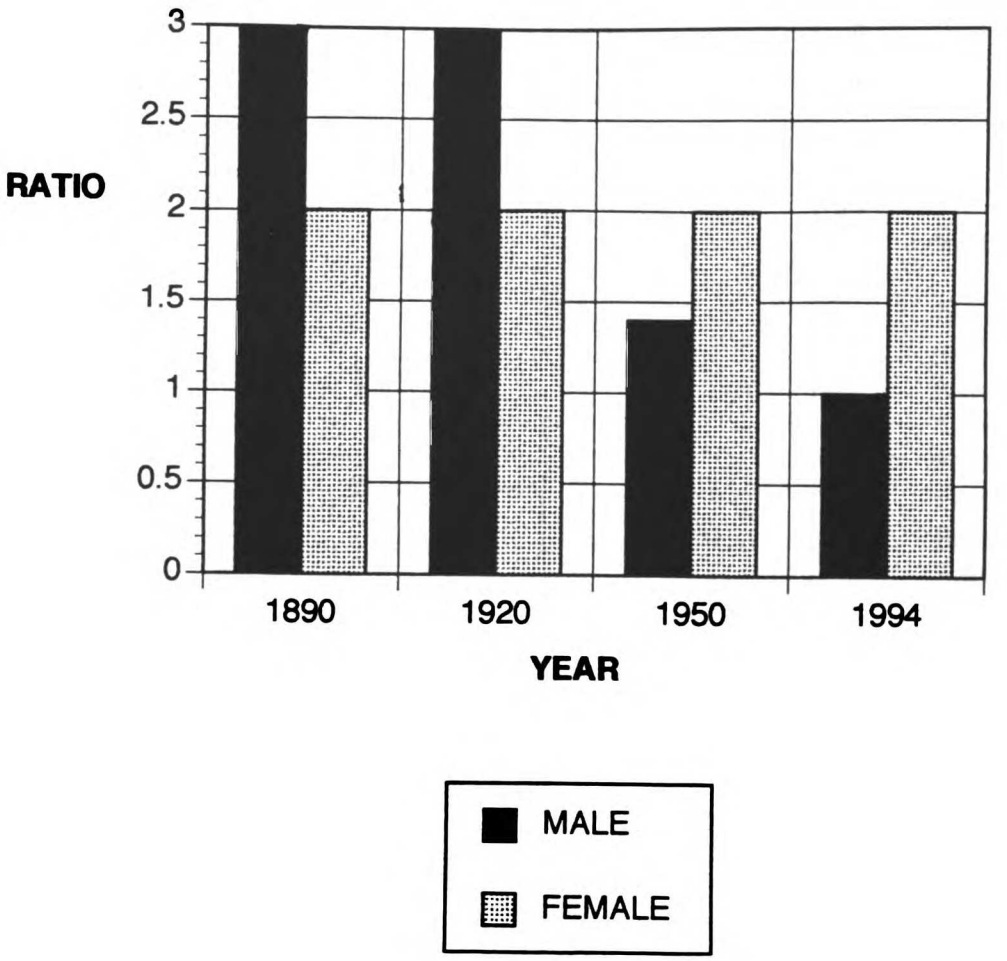
RATIO OF MALE TO FEMALE MULTIPLE SCLEROTICS (USA)

STUDY	Number of Patients MALE	FEMALE
Jelliffe	68	41
Stieglitz	17	17
Sachs	10	5
Moran	4	4
Montefiore Hospital	26	23
Vanderbilt Clinic	36	19
Mt. Sinai Hospital	55	38
	216	147
	57%	43%

Source: Association for Research in Nervous and Mental Diseases, *Multiple Sclerosis* (New York: Paul B. Hoeber, 1922), 36.

TABLE VI

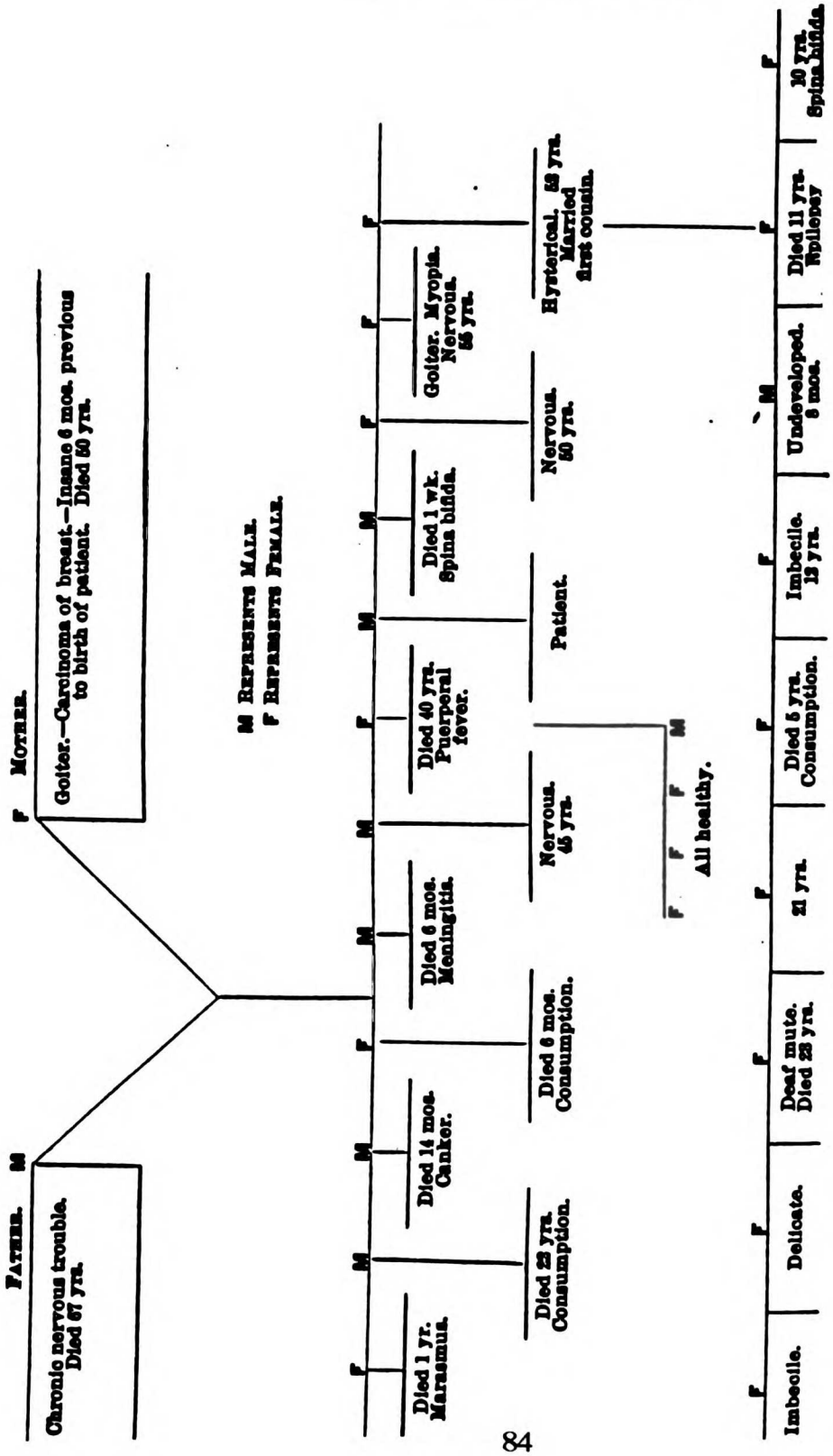
CHANGING RATIO OF MALE TO FEMALE MULTIPLE SCLEROTICS



SOURCE: Douglas MacAlpine, *Multiple Sclerosis* (Edinburgh: E. & S. Livingstone, Ltd., 1955). Association for Research in Nervous and Mental Diseases, *Multiple Sclerosis* (New York: Paul B. Hoeber, 1922). W.I. McDonald, "Multiple Sclerosis," in *Cambridge World History of Human Disease* (New York: Cambridge University Press, 1993), 883-887. Lawrence Steinman, "Autoimmune Disease," *Scientific American* (September 1993).

TABLE VII

TAINTED HEREDITY CHART



SOURCE:

Irwin H. Neff and Theophil Klingmann. "A Case of Multiple Cerbro-Spinal Sclerosis of a Special Anatomical Form, with a History of Pronounced Family Defect," *American Journal of Insanity* 56 (1899): 431-442.

TABLE VIII

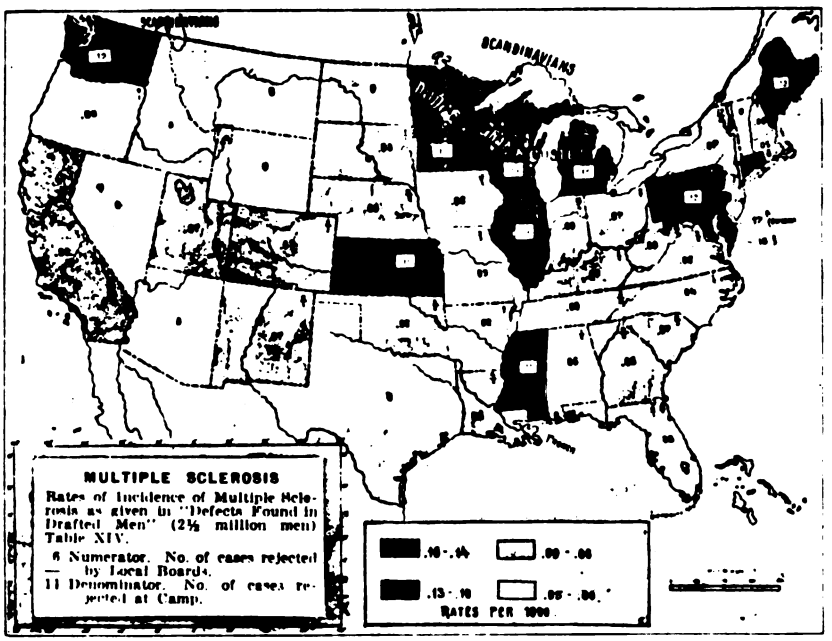
SHOWING THE OCCUPATION IN 110 CASES OF DISSEMINATED SCLEROSIS

Baker	2
Blacksmith	2
Bookkeeper	1
Brassfinisher	1
Canvasser	1
Chemical Worker	1
Clergyman	1
Clerk	4
Commercial traveller	1
Cook	1
Crofter	1
Dentist	1
Domestic Servant	1
Draper	2
Dressmaker	3
Dye worker	1
Rope works	1
Factory worker	1
Farmer	2
Fitter	2
Gardener	1
Grocer	3
Groom	1
Hat manufacturer	1
Hawker	1
Lives at Home (no occupation)	27
Housewife	16
Kitchen porter	1
Labourer	2
Lady's companion	3
Laundress	1
Map-engraver	2
Merchant	1
Miner	1
Music-teacher	2
Nurse, hospital	1
No occupation	1
Plumber	3
Pupil-teacher	2
School-girl	1
Shipping agent	1
Ship-owner	1
Shop-keeper	1
Wool-sorter	1
Not stated	4

SOURCE:
Byrom Bramwell, "On Disseminated Sclerosis, with Special Reference to the Frequency and Etiology of the Disease," Clinical Studies 2 (1904): 193-204

TABLE IX

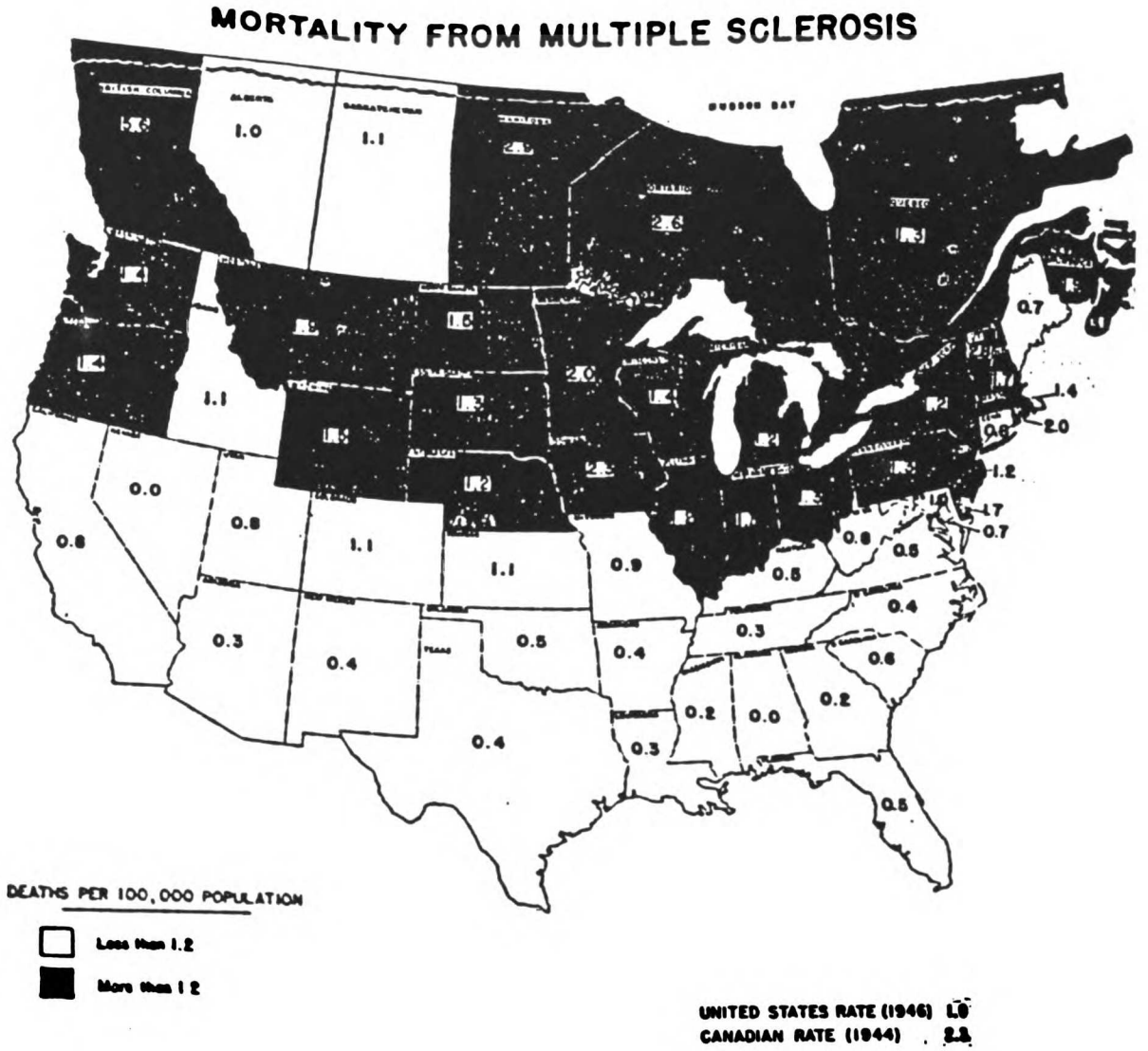
RATES OF INCIDENCE OF MULTIPLE SCLEROSIS
FROM DEFECTS IN DRAFTED MEN, WWI
(1917-1919)



SOURCE: Association for Research in Nervous and Mental Diseases, *Multiple Sclerosis* (New York: Paul B. Hoeber, 1922), 9.

TABLE X

FREQUENCY AND DISTRIBUTION OF MULTIPLE SCLEROSIS
(1944-1946)



SOURCE: Leonard T. Kurland, "The Frequency and Geographic Distribution of Multiple Sclerosis as Indicated by Mortality and Morbidity Surveys in the United States and Canada," Ph.D. diss., Johns Hopkins University, 1951.

TABLE XI

COURSE AND PROGNOSIS

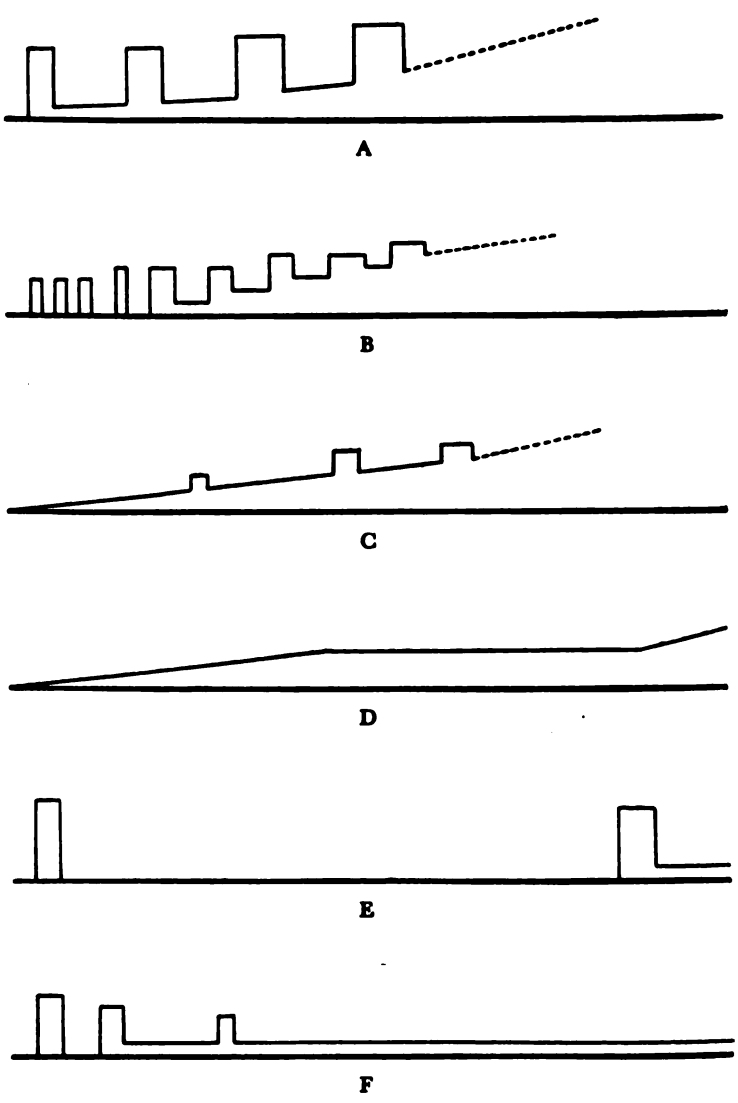


FIG. 7.

The course of multiple sclerosis

A, Relapses with early and increasing disability.
B, Many short attacks, tending to increase in duration and severity.
C, Slow progression from onset, superimposed relapses, and increasing disability.
D, Slow progression from onset without relapses.
E, Abrupt onset with good remission followed by long latent phase.
F, Relapses of diminishing frequency and severity; slight residual disability only.

SOURCE: Douglas MacAlpine, *Multiple Sclerosis* (Edinburgh: E. & S. Livingstone, Ltd., 1955), 147.