Title
A history of multiple sclerosis and medicine in the United States, 1870-1960

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Publication Date
1998

Peer reviewed|Thesis/dissertation

A History of Multiple Sclerosis and Medicine in the United States, 1870-1960

by

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DISSERTATION

Submitted in partial satisfaction of the requirements for the degree of

DOCTOR OF PHILOSOPHY

in

History of Health Sciences

in the

GRADUATE DIVISION

of the

UNIVERSITY OF CALIFORNIA

San Francisco

. . . . . .

Date **University Librarian**

Degree Conferred: . . . . . .

Dedication

This dissertation is dedicated to my grandparents, Gaylord Lee "Bill" Talley, Melba Lorene Talley, Margie I. Tucker, and Leon Tucker.

Acknowledgements

I would like to thank my dissertation committee chair Guenter B. Risse and my dissertation committee members David Hollinger and Lawrence Cohen for their help in the preparation of this manuscript. The late Jack Pressman guided my Master's Thesis on multiple sclerosis and encouraged me during my additional dissertation research. I would also like to thank Adele Clarke and Howard Kushner for reading and commenting on chapter one.

This dissertation was made possible by generous fellowship support by the University of California, San Francisco. This assistance included: a President's Research Fellowship in the Humanities for 1993/94 and 1996/97, a Regents' Research Fellowship in the Humanities for 1994/95 and 1995/96, a Graduate Dean's/Anthony Fee Scholarship for 1997/98, and a Graduate Division Research Award in 1996. The Department of the History of Health Sciences was very generous with Summer Research Fellowships in 1994, 1995, 1996, and 1997. I also received a Rockefeller Archive Research Grant in 1997 which made chapter four possible. In addition, the Graduate Student Association at the University of California, San Francisco provided money to present portions of this research at conferences in 1995 and 1997. For all this financial support I am deeply grateful.

In addition I would like to thank Katherine Donahue and Teresa G. Johnson of the Louise M. Darling Biomedical Library, History and Special Collections Division, University of California, Los Angeles for their help with the Putnam Collection. I would also like to thank the University of California, Los Angeles Medical Center Record Department for allowing me access to their hospital records. Adele Lerner gave valuable assistance at the New York Hospital/Cornell Archives.

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Abstract

This dissertation is the first history of multiple sclerosis (MS) in the United States. This study subjected the archives of the National Institutes of Health, the Commonwealth Fund, the American Neurological Association, the New York Academy of Medicine, and the New York Hospital, and three sets of patient records together with medical and popular literature to cultural and critical analysis.

Chapter one addresses how and why multiple sclerosis, as a disease category, emerged in a particular place and time. It emphasizes the importance of local conditions in the construction of nosological categories. Chapter two asks how and why MS went from being considered a rare disease in the late nineteenth century to one of the most common diseases of the central nervous system by the 1930s. It shows the importance of analyzing the history of individual diseases in the context of the larger ecology of diseases in which particular maladies are embedded. Chapter three examines the way that American culture conditioned research on the causes of MS in the late nineteenth and early twentieth centuries. It also shows the ways neurologists used MS for personal and institutional goals in addition to serving the needs of the still-forming specialty of neurology and how a particular model of funding laboratory research shaped the professional culture of biomedical research in the 1920s and 1930s. Chapter four analyzes how lay activists of the National Multiple Sclerosis Society made MS a popular crusade and a research priority in the late 1940s and 1950s. It shows the how the changes in American culture after 1945 created the conditions for an effective patient movement. It also elucidates a peculiarly American model of voluntary health movement activism. Chapter five analyzes the interaction of patients and physicians in the clinic through study of treatments for MS in the

1940s and 1950s. Most physicians adopted an attitude of therapeutic activism toward MS. This chapter illuminates this medical culture of therapeutic practice and the way patients impacted medical decision making.

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Introduction

One theme of this study is the different ways in which multiple sclerosis (MS) emerged into professional and popular consciousness as a disease problem in the United States. MS was born as a nosological category in France between 1862 and 1872. American neurologists transported this new intellectual category to the United States in 1870. The second pulse of emergence was a long one in which American neurologists gradually resignified MS from being a rare to a common disease from the 1870s to the 1930s as they more frequently diagnosed the condition. However, awareness of MS remained restricted to medical and mostly to neurological practitioners and was virtually unknown to laypersons. In the late 1940s and 1950s MS became well-known in popular culture as well. MS emerged as a popular crusade and a research priority in America as a result of the work of lay health activists who pressured the federal government, the National Institutes of Health, and neurological organizations to fund and conduct work on MS. Another main theme of this study is the manner in which the history of MS is inseparable from the history of neurology as specialty in the United States. In addition, MS has proven to be a useful probe in understanding the professional and popular cultures of health, medicine, and disease in the United States from the 1870s to the 1950s.

This study is based on cultural and critical analysis of multiple sources; these include: the archives of the National Institutes of Health, the Commonwealth Fund, the American Neurological Association, the New York Academy of Medicine, and the New York Hospital; three sets of patient records including: those of the New York Hospital; the University of California, Los Angeles Hospital; and those of a private neurologist named Tracy Jackson Putnam from Beverly Hills, California; medical and popular literature; conference proceedings; and one oral history.

Chapter one addresses how and why multiple sclerosis, as a disease category,

emerged in a particular place and time. It emphasizes the importance of local conditions, specialty structures, and styles of medical practice in the construction of nosological categories. Chapter two examines what happened to the category multiple sclerosis once neurologists transported it to the United States after 1870 in terms of diagnosis. It specifically addresses the question of how multiple sclerosis went from being considered a rare disease in the late nineteenth century to one of the most common diseases of the central nervous system by the 1930s. It shows the importance of analyzing the history of individual diseases in the context of the larger ecology of diseases in which particular maladies are embedded. It also shows the importance of professional and social factors in explaining the waxing of diseases. Chapter three examines the way that American culture conditioned research on the causes of MS in the late nineteenth and early twentieth centuries. It also demonstrates the ways neurologists used MS for personal and institutional goals in addition to serving the needs of the still-forming specialty of neurology. In addition, chapter three illuminates how a particular model of funding laboratory research shaped the professional culture of biomedical research in the 1920s and 1930s. Chapter four analyzes how lay activists of the National Multiple Sclerosis Society made MS a popular crusade and a research priority in the late 1940s and 1950s. It shows how the changes in American culture after World War II created the conditions for an effective patient movement. It also illuminates in a particular example, a peculiarly American model of voluntary health movement activism and the way American culture created a script for the sick role of the MS patient. In addition, it analyzes the relationship of the National Multiple Sclerosis Society to the National Institute of Neurological Diseases and Blindness in the 1950s. Chapter five analyzes the interaction of patients and physicians in the clinic through study of treatments for MS in the 1940s and 1950s. A popular culture of healing and a professional culture of medicine encouraged many physicians to adopt an attitude of therapeutic activism toward MS. This chapter elucidates

Chapter One
The Emergence of Multiple Sclerosis as a Disease Category in France and the United States, 1862-1882

I Introduction

Historians have employed six basic approaches in attempting to explain how and why disease categories emerge in specific times and places which are not necessarily mutually exclusive. The first style of reading the past comes from within the medical profession. Once physicians construct a new disease category they frequently reinterpret the medical past in an attempt to show that the "disease" had always been there after all. Retrospectively diagnosing cases is a cultural act of ahistorical appropriation which usually justifies physicians' current intellectual framework whether it is from the 1890s looking back or the 1930s looking back. To paraphrase Christopher Lawrence, a new disease category's existence is grounded in its having a past.[1] Roy Porter has argued that one reason physicians have been so preoccupied with retrospective diagnoses is that "for medical science it has been crucial to establish the historical stability of illnesses, physical and psychiatric alike" as part of its philosophic creed of concerning the essential nature of diseases and the nature of medical progress. "For if it can be shown through case analysis that patients in former centuries were definitely suffering from diseases presenting nowadays, the logical inference would be that such disorders display a natural incidence relatively unconstrained by socio-cultural determinants and independent of observer prejudice." This can serve professional and political interests: for instance, to refute the radically socially constructivist claims of the 1960s anti-psychiatry movement.[2]

A second approach has emphasized the role of new material conditions in the making of new disease categories. For example, Peter English studied the emergence of the new disease category of rheumatic fever in the nineteenth century out of the late eighteenth-century category of rheumatism. He argued that, in part, the emergence could be

explained by a change in virulence of the streptococcus involved in the disease. He thought that "a change in the streptococcus induced a host response that damaged the heart" in new ways in the early nineteenth century. Thus, physicians constructed a new disease category, rheumatic fever, centered on the heart. The important point, regardless if English is correct about rheumatic fever, is that biology does not stand still. Viruses mutate and organisms adapt. Therefore, the biological material conditions nineteenth-century physicians saw was not necessarily the same as what eighteenth-century physicians observed.

A third approach has emphasized the role of sociological structures in the generation of new disease categories. For example, Christopher Lawrence demonstrated that the category of coronary thrombosis emerged in the 1920s as a result of the construction of the new medical specialty of cardiology.[3] Another example would be the ways the changed structure of the hospital in Paris in the early nineteenth century, the Paris Clinic, led to the grounding of disease identities in pathological anatomy rather than in symptomatology.[4]

A fourth approach has stressed the importance of new ways and sites of practice in transforming the identity of a disease. For example, Andrew Cunningham has argued that there was a radical discontinuity between the identity of the new plague and that of the old plague. Physicians defined the new plague much more narrowly after 1894 when researchers proved *yersinia pestis* as the microbial actor responsible for plague, as we know it. That is, after 1894 the plague's identity depended exclusively on laboratory proof of the presence of the offending organism. Before the rise of the laboratory physicians constructed plague's identity through reference to symptomatology. Thus, the boundaries of what constituted "plague" in earlier times were much more fluid than after 1894. Therefore, for Cunningham, historians cannot reliably assume that "plague" in the past was the same as "plague" in the present.[5]

A fifth approach has examined the "social construction" of disease categories.

Cultural historians have analyzed the emergence and disappearance of disease categories such as: neurasthenia, chlorosis, homosexuality, drug addiction and alcoholism, masturbation, anorexia nervosa, hysteria, and chronic fatigue syndrome.[6] The strength of this approach is that it demonstrates Charles Rosenberg's point that "a disease does not exist as a social phenomenon until we agree that it does--until it is named."[7] The weakness of this approach is that it has centered on conditions where no one has shown an underlying pathophysiological mechanism. This obviously raises the question to what extent are diseases where there are identifiable pathophysiologies constructions? In other words, does the peculiar biology of a "disease" limit the interpretive possibilities of that condition?

Finally, a sixth approach, explicitly questioning radically constructionist position, has emphasized the interaction of cultural practices and biological events. Charles Rosenberg has posited the following historicist model of the process of disease definition: first, a "disease must be construed as a biological event little modified by the particular context in which it occurs." Physicians and patients, bound by culture and historical location, decide that a given biological event is pathological. Physicians and patients then respond to this biological event with the intellectual concepts of their time and place. For example, ancient healers used metaphors of cooking to explain the body's metabolism and derangements in ancient times; to physicians in the eighteenth and early nineteenth centuries, humoral models provided the explanatory framework for disease; in the early and middle nineteenth century, pathological anatomy provided a ground for the identity of diseases; the germ theory of disease provided an entirely different framework and basis for understanding various biological phenomena in the late nineteenth century; and today, hypothetical autoimmune mechanisms "are often used to explain diffuse chronic symptoms."[8] The strength of this historicist model is its emphasis on the role of the

mentalité of practitioners in generating new disease categories. Its weakness is that it is less able to explain historical change and its relatively static and unproblematic conception of biological events to be "framed."

Neurologists have used the first two approaches to explain the emergence of multiple sclerosis as a disease category. In this chapter I analyze these two explanations and find them unsustainable. I then offer a new explanation that emphasizes the importance of particular institutional structures and styles of practice in the generation of the new disease category, multiple sclerosis, in France and the United States, from 1862 to 1882.

II A Neurological Tale Of Discovery

Anglophone neurologists have told one another a story about the discovery of multiple sclerosis in essentially the same form from 1870 to 1993. One finds the story in the form of a prologue to neurological textbooks and published in proceedings of symposia on multiple sclerosis and occasionally as an article in a neurology journal.[9] G.E. Berrios and Ji Quemada have observed that this "view on how MS was 'discovered' became official by the end of the 19th century . . . The received view was consolidated by repetition in all the textbooks of the period."[10] This "official" story takes the form of the first approach mentioned above. That is, neurologists retrospectively diagnose cases and anatomical drawings as early instances of multiple sclerosis. In the process they divorce the cases from the intellectual context of the examples' historical period and give the extracted examples meanings quite different from those given them by past actors. In general, five organizing principles animate the retelling of this narrative: 1) is that the story is organized as a tale of steady progress in which one fact is simply adduced to another; 2) the narrative is concerned, as is usual in this genre, with the priority of discovery by notable individuals; 3) writers divorce physicians from their cultural, social, and intellectual contexts; 4) authors fictionalize connections between various past actors; and 5)

neurologists make retrospective diagnoses based on case histories in the medical literature and diaries.

The story usually begins with a discussion of two drawings from the 1830s by the pathological anatomists Jean Cruveilhier and Robert Carswell. G.E. Berrios and Ji Quemada have remarked that "whether the hero was Carswell or Cruveilhier depended on what side of the channel the writer was on."[11] As the French version of the story goes: "the earliest description of disseminated cerebro-spinal sclerosis is by Cruveilhier, in his *Atlas Pathologique du Corps Humain* (Paris, 1835-1842)."[12] Anglophone neurologists tend to give priority to the Scotsman Robert Carswell; American neurosurgeon and neurologist Tracy Jackson Putnam, writing in 1938, said that "the first pathologic specimen in a case of multiple sclerosis was described and illustrated by Sir Robert Carswell, professor of pathologic anatomy at the University College, London, in an atlas published serially up to 1838."[13]

As the story goes, following Carswell and Cruveilhier, "interest in the differentiation of this type from various other cord diseases, notably tabes and syringomyelia, now went on apace."[14] This line of research supposedly led to the Frerich's 1849 diagnoses of spinal sclerosis in several cases which were later confirmed by his student Valentiner in 1856. Also in 1856, Karl Rokitansky published a study on the new growth of connective tissue in the central nervous system associated with paraplegia. "In 1863 Rindfleisch gave an account of the pathology of the disease. He emphasized changes around vessels and in nerve elements; these he considered were due to a recurring or chronic inflammatory condition."[15] These German studies led to Jean Martin Charcot's celebrated work, "*Histologie de la sclérose en plaques*," (1868) which fully described the histological characteristics of multiple sclerosis using the then new staining techniques. By 1872 Charcot had precisely correlated these anatomical findings with clinical

symptomatology giving us his classic pathognomonic triad: nystagmus, scanning speech, and intention tremor.

The problem is that this linear story of progress ignores the historical context and meaning of anatomical drawings, physiological ideas, and clinical observations. Analysis of key elements of the narrative described above demonstrates the "official" story's inadequacies.

As mentioned above, writers usually cite Cruveilhier and Carswell as the two works showing the earliest illustrations of the sclerotic patches. Carswell studied with Pierre Charles Alexandre Louis in Paris during the height of the Paris Clinic. In 1831 he assumed the Chair of Pathologic Anatomy at University College, London where publishers commissioned him to make a collection of pathological drawings based on his experience in Paris.[16] In 1838 his *Pathologic Anatomy* appeared. In *Fasciculus Tenth, Atrophy*, with respect to Plate IV, figure 4, Carswell wrote that:

> the anterior surface of the spinal cord presented a number of spots, from a quarter of an inch to half an inch in breadth, of an irregular form, of a yellowish brown colour, smooth, glossy, without vascularity or any alteration in the colour or consistence of the surrounding medullary substance. The medullary substance thus affected was very firm, somewhat transparent, and atrophied. At the root of the medulla oblongata, these changes occupied the whole breadth of both the medullary fasciculi to the extent of half an inch in breadth from above downwards. Further down, they were confined to distinct spots on each fasciculus, and several of the same kind, but smaller, occupied the pons Varolii. The depth to which the medullary substance was affected in this manner varied from half a line to three or four lines, and on dividing the cord, it was seen to penetrate as far as the grey substance.[17]

Carswell understood this drawing through the then existent nosological category of atrophy *not* multiple sclerosis which did not yet exist as a medical category. His clinical note attached to the case shows that he intended no new nosological or clinical category. Carswell wrote that "I have met with two cases of a remarkable lesion of the spinal cord accompanied with atrophy. One of the patients was under the care of Mons. Louis, in the

Hospital of La Pitié, the other under the care of Mons. Chomel, in the Hospital of La Charité, both of them affected with paralysis. I did not see either of the patients, but I could not ascertain that there was any thing in the character of the paralysis or the history of the cases, calculated to throw any light on the nature of the lesion found in the spinal cord."[18] One of these patients was represented in Plate IV, figure 4, described above.[19]

Contemporaneously, though perhaps slightly later than Carswell, Cruveilhier illustrated lesions found on the pons and spinal cord of a thirty-seven year old woman during autopsy.[20] Writers from 1870 to 1993 have excised this particular case from its intellectual context and ascribed to it a significance different from what Cruveilhier stated. The clinical description and autopsy of *Dargès*, a female cook, who was at the *Salpêtrière* for two years before her death, appears in Cruveilhier's thirty-second *livraison* (book section) entitled: "*Maladies de la Moelle épinière: Etudes sur la paraplegie, paraplégie par compression de la moelle,*" (Diseases of the Spinal Cord: Studies of paraplegia, paraplegia by compression of the spine). The case of *Dargès*, sat between two other cases which Cruveilhier grouped together for the specific reason of exploring the localization of sensation and movement in the spinal cord and brain. The case before *Dargès*, was one of, "*Paraplégie complete du sentiment et incompléte du mouvement- Dégéneration grise des cordons postérieurs de la moelle,*" (Complete paraplegia of sensation and incomplete of movement- Gray degeneration in the posterior cords of the spine).[21] The *Dargès* case, the one often cited as being of the nosological category multiple sclerosis, was entitled "*Paraplégie-Dégéneration grise de la moelle, du bulbe, de la protubérance, des pédoncules cérébelleux, des couches optique des corps calleux, de la voûte à trois piliers* (fig. 4, pl. 2)," (Paraplegia- Gray degeneration in the spine, of the bulb, of the protuberance, of the cerebellar peduncles, optic thalami, corpus callosum and fornix). The third case was of, "*Paraplégie complète du sentiment et seulement incomplète de mouvement- transformation*

grise-jaunâtre des cordons posterieurs de la moelle." (Complete paraplegia of sensation and only incomplete of movement--gray-yellowish transformation of the posterior cords of the spine.)[22] Cruveilhier summed up the significance of the three cases as follows: "in the preceding case <the third>, there was paralysis of sensation and of movement; but the paralysis had gone farther in sensation than in movement; in the same way, in the other case, we saw the gray degeneration of the medial posterior cords affecting more the movement than the sensation. Still, the fact remains the same in this case, that we are able with rigor to claim as probable the localization of the faculties of sensation and movement. One is not able to conclude that the posterior medial cords preside exclusively over sensation."[23] Cruveilhier was interested in localization of the functions of sensation and movement as were most investigators of the spine in the 1830s and 1840s.[24] He did *not* construct a new disease category, multiple sclerosis; instead, he subsumed, what is usually seen as the critical case, that of *Dargès*, in the older category of paraplegia. Some writers take Cruveilhier's work out of its intellectual context and ascribe to it meanings from their own period whether it is 1870 or 1995.[25]

Cruveilhier and Carswell did not know of each other's drawings on what was perhaps demyelinated tissue and physicians forgot, not their whole atlases, but these specific drawings for at least fifteen years and maybe longer. Anthanassio Démosthène in his medical thesis from 1872 wrote that after Carswell: "for many years, no one occupied himself with this work and the question seems to have been forgotten . . ."[26] Jean Martin Charcot in his, *Leçons sur les Maladies du Système Nerveux faites à la Salpêtrière* (1872-73), described the contribution of Cruveilhier but added that from the 1830s to the 1855 this question seems almost entirely to have been forgotten.[27] Charcot did not cite Cruveilhier's work from the 1830s in his seminal histopathological study, "Histologie de la sclérose en plaques,"(1868).[28] Perhaps the Charcotians remembered or rediscovered this

one particular drawing from the 1830s between 1868 and 1870. Regardless, there was no linear research tradition connecting Carswell, Cruveilhier, and the relevant German researchers of the 1850s.

German physicians made several studies of spinal scleroses from 1849 to 1856. However, what these Germans meant by sclerosis, following Virchow's formulation, and what Charcot would mean by it were different.[29] For Virchow sclerosis meant thickening with condensation. During the 1860s what was meant by sclerosis became much more limited and precise for the French. On these grounds, French writers in the 1860s criticized German work on sclerosis for conflating many different pathological conditions in the clinic and in autopsy especially paralysis agitans with multiple sclerosis.[30] For example, in 1849 Frerichs of Breslau published a study giving a clinical diagnosis of spinal sclerosis but he did not separate out multiple sclerosis as a distinct nosological entity.[31] In 1856, Carl Rokitansky published a pathological study on the overgrowth of glial tissue in the central nervous system but correlated it with tabes dorsalis.[32] In 1856 Valentiner attempted to describe spinal sclerosis systematically but mixed together several different maladies according to Charcot.[33] In 1862 Skoda published a case in which, according to Charcot, "the diagnosis, paralysis agitans, had been made during life, and that, at the autopsy, they found patches of sclerosis scattered over all parts of the cerebro-spinal axis."[34] The German work did not establish a new nosological category. In the sense that a disease does not exist as a social phenomenon until it is named and given assent, multiple sclerosis did not exist until the 1860s when the famed French neurologist Jean Martin Charcot established the modern histopathological and clinical features of the disease.[35]

The problem is that Charcot did not work alone. Charcot himself gave ample credit to his associate Vulpian, though Vulpian's name is often forgotten by writers as they construct the heroic individual Charcot.[36] Both were part of a larger French medical

community concerned with *sclérose en plaques disséminées* in the 1860s.[37] From 1862 to 1872 Charcot, Vulpian, and several French physicians associated with them at *La Salpêtrière* including, Jaccoud, Ordenstein, Bourneville, Guerardand, Liouville and C. Bouchard at the *Hôpital de la Charité* established the nosological category *sclérose en plaques disséminées* as an individual disease.[38] It was not done by one lone hero.

The French school provided a clear and minute histological description of the sclerotic patches combined with a precise clinical symptomatology. In terms of the general anatomical topography, Charcot taught that "sclerosis in scattered patches" invaded "the brain, the protuberance, the cerebellum, the bulb, as well as the spinal cord."[39] The gray sclerotic patches had irregular outlines but were clearly circumscribed and sharply defined themselves from adjoining structures. Charcot wrote that "sometimes isolated, sometimes confluent, these patches or spots, as you can easily ascertain, are disseminated without any apparent rule, and as if at random, over all points of the cord."[40] He outlined three forms of the disease: the spinal form; the cephalic or bulbar form; and the cerebro-spinal form. Charcot then described the microscopic anatomy of the sclerotic patches paying particular attention to the "conjunctive envelope, which surrounds these elements on every side."[41]

Charcot described the histologic technique used to uncover this microscopic pathological anatomy of the sclerotic patches in his *Histologie de la Sclérose en Plaques*, (1868):

> 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 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 axis cylinder takes the

color of carmine, and the myelin envelope resists completely its action.[42]

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.[43] Charcot described the key pathognomonic feature of the sclerotic patch: "the "indefinite persistence, so to speak, of a certain number of axis-cylinders in the centre of portions which have undergone in the highest degree, the fibrillar metamorphosis is, observe, a character which appears properly to belong to sclerosis in patches. It is certainly not observed, at least in the same degree, in the other varieties of grey induration, whether there is question of descending spinal sclerosis consequent upon lesion of the brain, or of that which, occupying primitively the posterior cord, is justly considered as the anatomical substratum of progressive locomotor ataxy."[44]

Charcot adumbrated the clinical symptomatology of disseminated sclerosis which corresponded to the underlying pathological anatomy and which would differentiate it from other maladies especially paralysis agitans, chorea, Freidrech's ataxia, locomotor ataxy, and progressive general paralysis. In the cerebro-spinal form of disseminated sclerosis intention tremor was the key pathognomonic sign which marked the presence of disseminated sclerosis rather than paralysis agitans which had a constant tremor.[45] This cerebro-spinal symptom of disturbed movement could serve to distinguish "multilocular sclerosis" from chorea. In multilocular sclerosis the "general direction of the movement persists in spite of the obstacles occasioned by the shocks of the trembling;" while in chorea "the general direction of the movement would, in the accomplishing of this same act, be disturbed from its commencement, by absolutely contradictory movements" which would "prevent the patient effecting his object."[46] Also in the chorea there might be sudden unexpected movements when the limbs are at rest without any "intervention of the

will." This was not observed in multilocular sclerosis.[47] Finally, in progressive locomotor ataxia (sclerosis of the posterior columns of the spinal cord), Charcot held that ataxic incoordination was not characterized by trembling or rhythmic shocks but "rather gestures, more or less irregular."[48]

In terms of "cephalic" symptoms which pointed to multilocular sclerosis, Charcot placed special importance on disturbances of speech, sight, and "intelligence," particularly: diplopia, amblyopia, nystagmus and scanning speech. Charcot taught that difficulties in articulation might also be seen in "progressive general paralysis" but that when interpreted in light of concomitant phenomena, the diagnosis of multilocular sclerosis would be apparent.[49] Charcot observed vertigo in three-fourths of his cases of multilocular sclerosis which was useful because vertigo was not found in locomotor ataxy or paralysis agitans. He maintained that there was a peculiar facies to multilocular sclerosis: "the glance is vague, uncertain; the lips are drooping, half open; the features are expressive of hebetude, sometimes even of stupor." He characterized multiple sclerotics as emotionally labile as well.[50] Charcot outlined the paretic state of the lower limbs as pathognomonic of multiple sclerosis. He taught paresis usually affected one limb first, then another and might remit several times. Gradually the paresis would turn into a state of muscular rigidity and there might be painful muscular contractions and spasms in later stages of the disease.[51] This cluster of clinical symptoms together with the histological description discussed above are usually what later neurological writers remember as Charcot's contribution to multiple sclerosis. What they rarely discuss is the historical and intellectual context of Charcot's conceptualization of multiple sclerosis as a disease characterized by chronic inflammation of the neuroglia. In 1881 Charcot maintained that sclerosis of the nerve-centers corresponded to one of the modes of a primary chronic inflammation. Or, as Meredith Clymer put it in 1870: "to Dr. Charcot, therefore, unquestionably belongs the credit of distinguishing this

affection from other paralytic disorders, and notably from paralysis agitans, recognizing its pathological individuality, and tracing its clinical history. He 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."[52]

Inflammation was a typical mid-nineteenth-century concept which expressed the diseased state of the organism.[53] So, for Charcot, multiple sclerosis was a new disease category but not one with utterly individualized boundaries as disease is characterized in the late twentieth century.

The following is another example of the difference between Charcot's conception of the disease and a late twentieth century conception of it: "sclerosis of the nervous centres" came in three types in the new French nosology of the 1860s: 1) Disseminated or Multiple (*sclérose en plaques disséminées*); 2) Fascicular (*la sclérose rubannée*); or 3) Annular (la *sclérose annulaire*).[54] Within the first type, disseminated or multiple sclerosis, neurologists categorized three forms: cerebro-spinal, cerebral, or spinal.[55] Today, we might describe the "variety of forms the disease assumes" as benign, relapsing-remitting, relapsing-progressive, or chronic-progressive.[56] Today the key concept defining the ontology of the disease is the morbid physiological process; in the 1860s and 1870s it was where the lesions were anatomically. What defined the disease was not its "chronic inflammatory" character, a trope used to describe many diseases in the 1860s and 1870s, but rather its specific pathological anatomy which corresponded to a certain cluster of clinical signs.

III An Alternative Materialist Explanation

Neurologists have also used the second general approach outlined above in explaining the emergence of MS as a disease category in the nineteenth century; that is,

new material conditions gave rise to the new disease category. For example, neurologists Sten Fredrikson and Slavenka Kam-Hansen have argued that multiple sclerosis was a new disease of the nineteenth century in the sense that the underlying process of demyelination, regardless of nosology, was new. They based this conclusion on two sets of anatomical drawings from the 1830s and the reading of a single diary from 1822.[57] This deduction is unwarranted because there was a radically different nosology covering nervous diseases in the early nineteenth-century. Patients suffering demyelination could have been placed in any of the following categories: general paralysis, paralysis partialis, paraplegia, hemiplegia, chronic myelitis, chronic encephalitis, chronic inflammation of the brain and spinal cord, rhythmic chorea, choreiform paralysis, paralysis agitans, or tabes dorsalis as writers in the 1860s and 1870s recognized.[58] People with sclerotic patches on their spinal cords and brains could have been out there before the 1830s but we have no way of knowing for sure given the radically different nosological conceptions of disease during these previous eras.

Another problem with identifying early cases has to do with the problem that the reorganization of medical practice in early nineteenth-century France presents. The French Revolution led to a reorganization of the Paris hospitals that 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.[59] New institutional technologies led to new readings of the body;[60] before the Paris Clinic physicians categorized diseases by grouping similar symptoms. One contribution of the Paris Clinic was that diseases became identified with identifiable lesions. Pathological anatomy became the ontological ground of disease categories. Therefore, bodies with demyelination could have been there before but not seen because of different institutional structures, different

styles of medical practice, and a different way of grouping and categorizing disease categories.[61] Thus, the first two approaches, that is, rediagnosing past cases or emphasizing new biological material conditions, are unsatisfactory answers to the question of how and why MS emerged as a disease category in the nineteenth century.

IV French, German, and American Institutional Structures

A better way to address this question begins with comparing the institutional structures and styles of practice of neurologists in France, Germany, and the United States from the 1850s to the 1870s when interested physicians were forming neurology as a distinct discipline in these countries. This will allow us to answer the question of why it was French neurologists and not German or American ones who were able to establish the new disease category. The reason that Charcot and the French school were able to initiate the beginnings of what we consider the modern understanding of *sclérose en plaques disséminées*, rather than the Germans, had to do with the differences between the two groups with respect to the relationship between the clinic and the autopsy room.[62] The Germans were interested in the question of spinal sclerosis ten years before Charcot and had access to many of the laboratory tools Charcot did and this was a period of rapid growth in the field of pathological anatomy in Germany. Why then did the French triumph? Charcot named his German competitors writing 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."[63]

In the 1860s French and German physicians worked in parallel on the scleroses of the central nervous system. 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 *sclérose en plaques disséminées* specifically in the 1860s was partially rooted in Charcot's competition with the German research community over the fruits of the new staining techniques.[64]

However, the 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. Moreover, the German clinics were tiny in comparison to the French hospital clinics. This is a problem given what happens when a person suffers a demyelinating process in the central nervous system. The symptoms remit and relapse and the clinical signs are protean in presentation. At the *Salpêtrière*, Charcot and Vulpian had access to a stable group of patients who could be studied for an extended period of time and then autopsied.[65] Moreover, the *Salpêtrière* was huge. There were more than five thousand women at this hospice making the *Salpêtrière* virtually a village.[66] Charcot formalized his research method at the *Salpêtrière* as the *anatomo-clinique* approach. For this knowledge-producing system Charcot had reformulated Laennec's earlier *anatomo-pathologique* method by emphasizing close empirical longitudinal clinical study of chronic nervous diseases. Charcot also added the new German technology of microscopic histology which produced another layer of pathological information, that of cellular pathology, to gross pathological anatomy studies at autopsy.[67] Because of the fluid, decentralized, and smaller nature of the German medical institutions these combinations of long-term clinical observation, large numbers of patients, and autopsies were harder to achieve.[68]

For example consider the careers of four of the German researchers considered

early contributors to the study of *sclérose en plaques disséminées*. 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.[69] 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 assistant ordinarius professor in 1864. In 1865 he was named ordinarius professor of pathological anatomy in Bonn.[70] 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 to 1851 he was an assistant physician at a Leipzieg 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.[71] 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 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.[72]

Only in Paris did the institutional social structure allow the autopsy room and the clinic to be combined for a long enough period of time so that Charcot and others could describe both the pathological lesions and the clinical symptoms in exhaustive detail.

Charcot was in the third generation of Parisian physicians who practiced the anatomical/clinical synthesis which combined clinical and surgical approaches and shifted power from patients to physicians. This synthesis revolutionized what constituted the identity of a disease. This style of practice shifted the identity of disease from being grounded in symptomatology to pathological anatomy. The late eighteenth-century natural history conception of disease gave way in the early and middle nineteenth century to disease correlated with identifiable lesions. Guenter Risse has observed that French physicians practicing in huge institutions "turned from unreliable patient histories to pathological findings obtained under more controlled circumstances at the autopsy table" as a way of making sense out of the vast panoply of pathological symptoms encountered. "In a complete turnabout, pathology came to rule clinical medicine, narrowing its scope to those complaints which could be correlated to internal lesions."[73] The Germans did excellent work in their pathological laboratories but the researchers who looked at the scleroses in the laboratory did not have as stable a patient population, as the French did, to correlate the clinical symptomatology with the pathological laboratory.[74] Moreover, Charcot's method of close clinical observation followed by autopsy was a style of research explicitly in oppostion to the experimental physiology of François Magendie and Claude Bernard. Charcot believed that animal experimentation was not a promising method to solve neurological questions.[75] Thus the honors went to the Charcotian School which was able to carefully and systematically observe symptoms in the clinic and then go the autopsy room to explain what had been observed.

Sclérose en plaques disseminées also emerged through a subdivision of previous nosological categories in Europe and through the development of a more subtle descriptive clinical language. G.E. Berrios and Ji Quemada have written that " . . . the disease was disentangled on *descriptive grounds alone* <italics theirs>. It concerns the development of

an ever subtler language for the description of motor and sensory symptoms."[76] Once the medical category, disseminated, multiple, multilocular, cerebro-spinal sclerosis, or insular sclerosis (the five most common early anglophone translations) came into existence in the 1860s, physicians reread their own past through this new category and created a fictional story. Following generations received this story uncritically, rereading the past through the conceptual lens of their own times. Writers selected the earlier cases and studies in the literature for inclusion in the discovery narrative and divorced them from their original historical contexts. This served to fictionalize a tale of linearity and connect researchers and studies that had little or no relationship. A more accurate reading of the emergence of disseminated sclerosis in Europe is one that focuses on France where the confluence of a new histopathological technology, a unique relationship between patient and physicians in the clinic, a unique relationship between the clinic and the autopsy room, and a neurological culture reemphasizing disease specificity served to reorganize clinical perception and construct a new disease category.[77] Also, mid-nineteenth-century France saw the increasing subdivision of medical knowledge and the creation of the new medical specialty of neurology. "As a result of government support, hospitals and physicians increasingly focused on the study and treatment of subcategories of illness."[78] New professional structures and practices created new medical visions of diseased bodies.

V American Translations Of *Sclérose En Plaques*

American physicians interested in the nervous system were constructing the discipline of neurology in the United States in the 1870s. (See chapter two). However, the structure of medical practice in the United States during this time meant that the practice of neurology would be quite different.[79] American physicians had no equivalent to the large, captive patient populations of France, or the cultural authority of German physicians, or the

protection of the state in policing professional boundaries. Nevertheless, the structure of American medical practice does partly explain how and why the medical category of multiple sclerosis came to the United States through a process of translation and dissemination from 1870 to 1882. Americans had rare access to the autopsy room and they practiced on a highly mobile patient population marked by a more traditional power relationship between patients and physicians. The possibilities for making the kind of scientific contributions in neurology coming from France did not exist in the United States in the 1870s. Regardless, some American physicians were attempting to construct the discipline of neurology in the United States during this time period. On what basis could they do this? What practices would constitute neurology? On what grounds could American neurologists claim expertise? In the construction of the discipline of American neurology, reading and interpreting the latest neurological findings from Europe became a constitutive practice for American neurologists. Knowing the latest neurological disease categories, together with clinical diagnosis, were the markers of specialization with which American neurologists could claim expert knowledge and thus differentiate themselves from other physicians. Once multiple sclerosis entered the American medical lexicon, American physicians also reinterpreted past medical literature and reconstructed what they saw in the clinic and found cases of multiple sclerosis in the United States. For American neurologists this scholarly exercise was part of what made them neurologists, rather than general physicians, in the American context. By 1882 the reorganization of medical perception in the nervous clinic with reference to multiple sclerosis was mature and the category became naturalized. Multiple sclerosis emerged in the United States because of the reception and translation of a new French category and the subdivision and resignification of previous disease categories. American neurologists then reinterpreted the bodies in front of them as multiply sclerotic through the intellectual filter of the new disease category. These processes of translation and dissemination led to the reorganization of

medical perception in the American nervous clinic.

Multiple Sclerosis was not yet part of the American medical lexicon in the 1860s. One can see this through an analysis of three related cultural fields through time: medical dictionaries, textbooks, and journal articles. In medical dictionaries used in America from 1860 to 1868 the definition of sclerosis still signified the older meaning of Virchow: "thickening with condensation."[80] In C. Handfield Jones' textbook, *Clinical Observations on Functional Nervous Disorders* (1868), disseminated sclerosis does not appear.[81] Dr. J.C. Morris presented the "case of the late Dr. C.W. Pennock" with autopsy in July 1868 in the *American Journal of Medical Sciences*.[82] This case would *later* be retrospectively diagnosed as one of multiple cerbebro-spinal sclerosis but it was *not* so categorized during the patient's life *nor* at the autopsy performed by Dr. John H. Packard on April 18, 1867 *nor* at the microscopical examination done by S. Weir Mitchell.[83] Another article later reinterpreted as multiple sclerosis several years after it originally appeared was attributed to M. Gonzalez Echeverria. The publication was the result of F. A. Castle's student notes. The study was entitled "Sclerosis of Both Third Anterior Frontal Convolutions Without Aphasia" and appeared March 1, 1869.[84] In it Echeverria reported an autopsy wherein, "the brain tissue was in a general state of sclerosis, arrived to its highest degree in the superior and inferior marginal convolutions and along the fissure of Sylvius on either side. Under the microscope, the nervous elements appeared deficient, and replaced by a multiplication of connective cells and fibres . . . As to the nerves connected with eruptive patches they had undergone a fatty degeneration of their primitive fibres, with the same rank growth of connective elements."[85]

Nowhere in the article does Echeverria connect this with the seminal work being done in France at the time nor does he categorize it as a new disease category; though, one can see the grounds on which later readers reinterpreted the article.[86] In C. Handfield

Jones's textbook, *Clinical Observations on Functional Nervous Disorders* (1868), disseminated or multiple sclerosis does not appear.

Philadelphia Professor Meredith Clymer first translated the disease category, *sclérose en plaques disséminées* into an American idiom in 1870. He rendered this new disease category as disseminated, diffuse, or multilocular sclerosis of the brain and spinal cord.[87] Clymer wrote: "to Dr. Charcot, therefore, unquestionably belongs the credit of distinguishing this affection from other paralytic disorders, and notably from paralysis agitans, recognizing its pathological individuality, and tracing its clinical history."[88]

Clymer presented Charcot's views on pathological anatomy of disseminated sclerosis: "in the central zone, that is, in the midst of the sclerosed patch . . . the axis-cylinders are atrophied to such a degree that it is hard to distinguish them from the newly-formed fibrils. The persistence of these cylinders in the midst of the tissue which has undergone fibroid substitution, is Dr. Charcot thinks, peculiar to disseminated sclerosis."[89]

Clymer then presented the Charcotian taxonomy of the three forms of disseminated sclerosis: the cerebral form, spinal form, and cerebro-spinal form. Clymer outlined the pathognomonic signs of ataxic gate, paresis, intention tremor, nystagmus, scanning speech, paresthesia, amblyopia, photopsia, muscular spasms and cramps and how to coordinate particular physiological symptoms with the anatomical location of particular lesions. He ended with the presentation of sixteen cases that illustrated each form of the disease; one was a retrospective diagnosis made from a case from the American medical literature; fifteen were translations of European cases. Clymer created no new knowledge about disseminated sclerosis but simply began the process of translating the new disease category into the United States.

Clymer acted as a filter through which the French work passed to American physicians nationally. See how Henry D. Noyes, Professor of Ophthalmology at Bellevue

Hospital Medical College and Surgeon to the New York Eye and Ear Infirmary, used Clymer's translated model to reinterpret a case. Noyes reported the case of Miss A. who suffered from diplopia, pallor of the optic discs, general muscular paresis, and an intention tremor. Noyes had diagnosed a "tumor within the skull" during the patient's life but, "when the brain was examined by two skilful <sic> pathologists <Dr. Francis Delafield and Dr. Eno>, they could, with the naked eye, detect nothing in it abnormal." Noyes lamented that "there was no examination of the brain-tissue under the microscope-nor was the cord examined . . . The unsatisfactory conclusion of this case gave me great disappointment. I was unable to account for its remarkable features despite the opportunity of an autopsy. In searching the literature <f>or light, I at length found what seems to me the true explanation of the case in the hypothesis of disseminated sclerosis of the brain and spinal cord to which Charcot called attention. An excellent account of this disease appeared in the *New York Medical Journal* for May and June, 1870, and in the *Medical Record* for August, 1870, by Dr. Meredith Clymer."[90] Noyes' reinterpretation of old cases based on textual evidence was an example of one of the key elements of neurological practice in the American context during the period of active translation of the European category into the American medical lexicon during the 1870s and early 1880s.

Regional medical journals offer evidence of the dissemination of the new disease category into the hinterlands. Physician C.H. Boardman of St. Paul, Minnesota, writing in *The Northwestern Medical and Surgical Journal* in 1873, taught how the provincial clinician might recognize progressive multiple cerebro-spinal sclerosis. Boardman followed Clymer explicitly citing the key symptoms of: scanning speech, nystagmus, intention tremor, weakness, paresthesia, and paresis as pathognomonic. Boardman offered no new cases of his own but reinterpreted an old case from the medical literature for an example. Boardman wrote that "it is not long since the diagnosis of hemiplegia or paraplegia was deemed sufficiently minute and accurate, and one of these terms or that of

myelitis was applied to a large proportion of cases of disease of the central nervous system. since the recognition of sclerotic palsy it is plain that the disease is of more common occurrence than was at first supposed: it has probably heretofore been confounded with paralysis agitans, myelitis or meningitis; again, it may simulate locomotor ataxy . . .The fullest definition of the disease is that given by Dr. Clymer . . ."[91]

Here is another example of this process of translation and dissemination of the new disease category to the provinces. J.K. Bauduy, Professor of Psychological Medicine and Diseases of the Nervous System at the Missouri Medical College, published a clinical lecture on "Multiple Cerebro-Spinal Sclerosis" in 1874. The article basically taught its readers about the French model and how to make a differential diagnosis of multiple sclerosis. Bauduy wrote that "until within comparatively a very recent period paralysis agitans, multiple cerebral sclerosis and multiple cerebro-spinal sclerosis were inextricably confounded together under the one common name of paralysis agitans . . .
Multiple Sclerosis . . . involves several parts of the same ganglion, and consists of nodules or plates of sclerosed tissue scattered throughout its substance . We are indebted to the comparatively recent researches of Messrs. Charcot and Vulpian, for the proper elucidation of this difficult subject, and Dr. Meredith Clymer of New York was the first to present their views, somewhat modified by his own opinions, to the American medical profession."[92]

Here is another example of the translation and dissemination of the new disease category to a local, but nationally important audience, the principal aim of which was to teach readers how to make the differential diagnosis between paralysis agitans, locomotor ataxia, and multiple sclerosis. George S. Gerhard, physician to the Orthopaedic Hospital and Infirmary for Nervous Diseases in Philadelphia, in "Cases of Multilocular Cerebro-Spinal Sclerosis" (1876), in the *Philadelphia Medical Times* remarked that "the cases reported above are well-marked examples of a very interesting and uncommon disease, and one which, singularly enough, was not minutely described until 1862, when Vulpian and

Charcot published a series of cases . . . the disease was not acknowledged to be a distinct one until after the appearance of the reports alluded to, and even then its recognition was entirely confined to France and Germany. This seems strange to us, now that we know that the disease has a definite lesion and is accompanied by a pretty consistent train of symptoms. It must be borne in mind, however, that from the nature of the lesion the symptoms known to be peculiar to the disease may be greatly modified by being mixed up with those belonging to other disorders of the cerebro-spinal axis."[93] He then basically laid out the French model of the disease.

The other native filter which medical readers and writers cited as a source of the new knowledge was William Hammond's textbook, *A Treatise on Diseases of the Nervous System*.[94] The first edition appeared in 1871 and the second in 1872. Hammond's translation came with significant reinterpretation of the French work and with marked differences from Clymer's rendition of the new disease category. Hammond's textbook listed, among others, two categories under diseases of the brain: diffused cerebral sclerosis and multiple cerebral sclerosis. Under diseases of the spinal cord he labeled the spinal form of multiple sclerosis as sclerosis of the antero-lateral columns of the spinal cord; he classified multiple cerebro-spinal sclerosis in the section on the cerebro-spinal diseases. Hammond cited the seminal French work: "it is only recently, mainly through the observations of Charcot and Vulpian, that attention has been again directed to sclerosis of the cerebro-spinal variety, a form which differs from those already described in this treatise, both in its extent and in the symptoms by which it is characterized."[95]

Hammond drew sharp differences between the cerebral, spinal, and cerebro-spinal forms of *sclérose en plaques disseminées*, to the extent that for Hammond they were virtually separate diseases. With regard to multiple *cerebral* sclerosis Hammond wrote that "age is certainly one of the most powerful predisposing causes of multiple cerebral

sclerosis mainly affecting the hemispheres, and causing the symptoms heretofore classed as paralysis agitans. Thus of nine cases in which I diagnosticated the disease in question, all were over fifty years of age, and three were over sixty."[96] Basically Hammond held that multiple cerebral sclerosis was what used to be called *paralysis agitans* by some authors which indicated that he had not either fully digested the French work or did not agree with it. Hammond admitted that "the whole subject is so confused in the minds of most authors that it is difficult to make out clearly what they refer to under the designation of paralysis agitans."[97] He recognized that he was alone in seeing multiple cerebral sclerosis as a separate disease. Hammond wrote that "the first question to be considered under this head relates to the existence of multiple cerebral sclerosis as an independent affection--that is, without lesions of like character being at the same time produced in the spinal cord. The weight of authority is probably against the view expressed in this chapter."[98] Hammond seems to have split what was *paralysis agitans* into two different diseases one of which was the "cerebral form" of multiple sclerosis.

Hammond rephrased the "spinal form" of disseminated sclerosis as: sclerosis of the antero-lateral columns of the spinal cord. In Charcot's work the axis cylinders were shown to be relatively spared after the destruction of the myelin sheath. Charcot considered this the pathognomonic key in terms of pathological anatomy. Hammond did not seem to understand this because he taught that the axis cylinders were annihilated in these varieties of multiple sclerosis.

Regardless of Hammond's alternative translation and misunderstanding he was another source for new the new disease category, at least cerebro-spinal sclerosis, especially for physicians in the provinces. For example, John I. Cook of Elizabethtown, Kentucky in an 1872 article published in *The Richmond and Louisville Medical Journal* held that with regard to multiple cerebro-spinal sclerosis "Professor Hammond, in his

valuable work, says he has seen nine cases only; and it is to him that the Profession is indebted for a clear and succinct description of the disease."[99] Michigan physician Stiles Kennedy in "Cerebro-Spinal Sclerosis, Involving the Hemispheres" published in the *Detroit Review of Medicine and Pharmacy* (1873) also credited Hammond as being his source for the new knowledge. However, Kennedy disputed Hammond's sharply drawn distinction between multiple cerebral sclerosis and multiple cerebro-spinal sclerosis.[100] Horatio C. Wood, Professor of Materia Medica and Therapeutics and Clinical Professor of Nervous Diseases, in a 1878 clinical lecture delivered at the Medical School of the University of Pennsylvania credited Hammond as the primary source of his lecture.[101]

Another textbook source might have been Allan Mclane Hamilton's, *Nervous Diseases: Their Description and Treatment* (1878). Hamilton, discussing cerebrospinal sclerosis, wrote that "for a long time this disease was mistaken for paralysis agitans (Parkinson's disease), chorea, and other neuroses; and even after it had been shown to be a separate neurosis a certain amount of confusion existed in regard to its nomenclature and its position among the scleroses. Charcot and Moxon are to be thanked especially for their successful efforts to give it a distinct character."[102] Hamilton then went on to discuss one case of his from two years before of a patient he saw "but once."[103] He translated a 1869 case from Bourneville and discussed a 1876 case from Dr. Geo S. Gerhard out of the *Philadelphia Medical Times*. He was teaching an American audience about the new disease category through textual analysis and textual reinterpretation of one of his own previous cases. This was, in fact, a large part of what constituted neurological practice in the United States at that time.

Another source of the new knowledge came from American translations of European textbooks. For example, in 1879 L. Putzel translated Viennese Professor M. Rosenthal's, *A Clinical Treatise on the Diseases of the Nervous System, with a preface by*

Professor Charcot, which included a chapter on,"Sclerosis of the Brain and Spinal Cord."[104] Rosenthal presented the microscopical examinations of Vulpian and Charcot to illustrate the pathological anatomy of the new disease. Some American physician's may have read the French works directly if they had access to foreign medical journals. For example, San Francisco Professor Joseph O. Hirschfelder gave a clinical lecture on disseminated sclerosis at the Medical College of the Pacific in 1882. He said that "this disease was first thoroughly investigated by the celebrated Charcot, who has taught us all of importance that we know of the disease, and the following remarks shall be based principally upon the results of his investigations."[105]

Another example of trans-Atlantic knowledge transfer comes from November 1877. A twenty-six year old man came to William Osler, Professor at the Institute of Medicine at McGill University and Physician to the Montreal General Hospital, complaining of trembling of the head and arms. Osler reported that "by a happy coincidence, he had just been reading a paper on multiple sclerosis in Zimssen's Archives. The symptoms of this patient corresponded so closely to those described in the paper that the diagnosis was clear."[106]

VI Conclusion

For American neurologists the disease category came first and served to reorganize clinical perception of what was really "there." Two practices grounded the nascent identity of neurology in the United States: one was the almost scholastic rereading and reinterpreting of old cases and the medical literature and the second was expert clinical diagnosis. In the next chapter we explore what happened to the category of multiple sclerosis in the United States in the 1880s and beyond especially in terms of diagnosis in the clinic. A key element in explaining the generation of the disease category multiple

sclerosis and its fate in the twentieth century was the rise of neurology as a medical specialty; however, what consituted neurology, its professional culture, its structures, and its practices depended on local conditions. The generation of the category of multiple sclerosis in Europe from 1862 to 1872 and its fate in the United States in the late nineteenth and twentieth centuries demonstrates this. Because neurological practice depended on local circumstances and because patients' experience of illness depended on particular cultural ways of being sick, the history of multiple sclerosis was different depending on the country studied.

ENDNOTES

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4. Michel Foucault, *The Birth of the Clinic: An Archaeology of Medical Perception*, trans. A.M. Sheridan Smith (New York: Pantheon Books, 1973). Russell Charles Maulitz, *Morbid Appearances: the Anatomy of Pathology in the Early Nineteenth Century* (New York: Cambridge University Press, 1987). John Harley Warner, "Remembering Paris: Memory and the American Disciples of French Medicine in the Nineteenth Century," *Bulletin History Medicine* 65 (1991): 301-325. Guenter B. Risse, "A Shift in Medical Epistemology: Clinical Diagnosis, 1770-1828," in *History of Diagnostics, Proceedings of the 9th International Symposium on the Comparative History of Medicine--East and West*,, ed. Yosio Kawakita (The Taniguchi Foundation 1984, Japan), 115.

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7. Rosenberg, "Introduction," *Framing Disease*, xiii.

8. Ibid., 211-19.

9. Examples of this include: Meredith Clymer, "Notes on the Physiology and Pathology of the Nervous System, with Reference to Clinical Medicine," *New York Medical Journal* 11 (1870): 225-260. Walter Timme, "Multiple Sclerosis--Historical Retrospect," in *Multiple Sclerosis [Disseminated Sclerosis] An Investigation by the Association for Research in Nervous and Mental Diseases, Report of the Papers and Discussions at the Meeting of the Association; New York City, December 27th and 28th, 1921*, ed. Charles L. Dana, Smith Ely Jelliffe, Henry Alsop Riley, Frederick Tilney, Walter Timme (New York: Paul B. Hoeber, 1922), 1-8. Tracy J. Putnam, "The Centenary of Multiple Sclerosis," *Archives of Neurology and Psychiatry* 40 (1938): 806-813. Walter Timme, "Multiple Sclerosis--Historical Retrospect," in *Multiple Sclerosis and the Demyelinating Diseases, Proceedings of the Association for Research in Nervous and Mental Diseases, December 10 and 11, 1948, New York, Volume XXVIII*, ed. Henry W. Woltman, H. Houston Merritt, S. Bernard Wortis, and Clarence C. Hare (Baltimore: Williams &Wilkins Company, 1950), 3-11. Douglas McAlpine, Nigel D. Compston, Charles E. Lumsden, "Chapter 1 Historical Note," in their *Multiple Sclerosis* (Edinburgh and London: E. & S. Livingstone, Ltd., 1955), 1-6. Allan L. Sherwin, "Multiple Sclerosis in Historical Perspective," *McGill Medical Journal* 26 (1957): 42-43. R. Medaer, "Does the history of multiple sclerosis go back as far as the 14th century," *Acta Neurologica Scandinavica* 60 (1979): 189-92. A. Compston, "The 150th anniversary of the first depiction of the lesions of multiple sclerosis," *Journal Neurology Neurosurgery Psychiatry* 51 (1988): 1249-1252. 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 (1989): 237-243. A. Compston, "The dissemination of multiple sclerosis," *Journal College Physicians London* (1990): 207-218. W.I. McDonald, "Multiple Sclerosis," in *Cambridge World History of Human Disease*, ed. Kenneth F. Kiple (New York: Cambridge University Press, 1993), 883-887. W.I. McDonald, "The dynamics of multiple sclerosis: The Charcot Lecture," *Journal of Neurology* 240 (1993): 28-36.

10. Berrios and Quemada, 177.

11. Ibid., 177-78.

12. Clymer, 228.

13. Putnam, 806. Exactly which drawing appeared first has remained of interest to neurologists into the 1990s but this concern with priority is misguided because, as will be shown, neither was concerned with or created a new nosological category, "multiple sclerosis." For the continuing concern with priority see McDonald, "The Dynamics of Multiple Sclerosis," 29. Compston, 1249-1252.

14. Timme, 1922, 4. Timme reprinted the article as an introduction to another conference held in 1948, the proceedings of which were published in 1950. Timme, 1950, 5. Timme correlates the wrong case history with the drawing usually seen as descriptive of multiple sclerosis. It was the thirty-seven year old cook whose autopsy produced the drawing not the fifty-four year old woman whose case Timme relates. This case came directly after the case of the thirty-seven year old cook.

15. McAlpine, 2.

16. Ibid., 1.

17. Robert Carswell, *Pathological Anatomy: Illustrations of Elementary Forms of Disease, Fasciculus Tenth, Atrophy* (London: Longman, Rees, Orme, Brown, Green, and Longman, 1836), 4.

18. Ibid., 4.

19. Ibid. Alastair Compston seems confused about the drawing in question for Carswell. He first cites plate 4, figure 1 on page 1249 and then he cites plate 4, figure 4 on page 1250. The correct figure and notes correspond to plate 4, figure 4. See Compston, 1249-50.

20. McDonald, "The dynamics of multiple sclerosis: The Charcot Lecture," 29.

21. Jean Cruveilhier, *Atlas Pathologique du Corps Humain, ou descriptons, avec figures lithographiées et coloriées des diverses altérations morbides dont le corps humain est susceptible; tome second; livraison 32* (Paris: Chez J.B. Baillière, 1835-1842), 21.

22. Ibid., 24.

23. "Réflexions. Dans le cas précédent, il y avait paralysie du sentiment et du mouvement; mais la paralysie avait beucoup plus porté sur le sentiment que sur le mouvment; de même que, dans d'autre cas, nous avons vu la dégéneration grise des cordons médians postérieurs affecter bien plus le mouvement que le sentiment. Toujours est-il que, même dans ce cas, qu'on pourrait à la rigeur invoquer comme favorable à la localization des facultés sensible et motrice. On ne peut pas conclure que les cordons médians posterieurs président exclusivement au sentiment," Cruveilhier, 24.

24. Berrios and Quemada, 176-177. For François Magendie's research on localizaton of sensation and movement in the spine see John E. Lesch, *Science and Medicine in France, 1790-1855* (Cambridge, MA: Harvard University Press, 1984), 166-96.

25. Michel Bonduelle says that "in contrast, the patients with predominant action tremor had the distinctive multifocal sclerotic lesions of the central nervous system that had been termed *'sclérose en taches ou en isles'* by Cruveilhier." Bonduelle cites the source of this as (Cruveilhier, 1829-42). Bonduelle does not narrow the cite down more than this. Cruveilhier's *Anatomie Pathologique* is composed of two huge volumes. The case of Dargès, often claimed to be one of multiple sclerosis, does not contain the phrase *"sclérose en taches ou en isles."* It is unclear from where Bonduelle draws this information. Christopher G. Goetz, Michel Bonduelle, and Toby Gelfand, *Charcot: Constructing Neurology* (New York: Oxford University Press, 1995), 115. See also Cruveilhier *Anatomie Pathologique, tome second, 32 livraision*, 22,23,24. Cruveilhier wrote about "isles de substance blanche" (p. 23,24). Cruveilhier wrote that "les filets nerveux qui naissent de bulbe, savoir, les racines du grand hypglosses, du glosso-pharyngien et du pneumogastrique, sont gris, au moins en apparence, réduits à leur névrilème" (p. 22).

Cruveilhier also wrote that "il m'a paru qu'il y avait seulement destruction de la couche blanche, qui revêt las substance gris" (p. 23). In general, on these gray transformations Cruveilhier wrote that "j'ai trouvé cette transformation grise avec induration sur le trajet de plusieurs radiations des corps striés. Reste à déterminer en quoi consiste cette transformations grise. On ne saurait ne pas admettre une transformation de tissu; car dans un grande nombre de points, les parties grises étaient fasciculées et représentaient parfaitement, sauf la couleur, les parties blanches qu'elles remplaçaient. De nouveaux faits viendront sans doute nous éclairer à cet régard" (p. 23).

26. "Pendant quelques années, on ne s'en occupe plus et la question paraît être tombée dans l'oubli, lorsqu'en 1848 Frerichs de Breslau publie de nouvelles observations sur la sclérose," Anthanassio Démosthène, *Contribution a L'étude de la Sclérose en Plaques dissémineés avec deux nouvelles observations*, Thèse, Montpellier le 14 décembre 1872, New York Academy of Medicine <hereafter NYAM>, 10-11.

27. 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. Berrios and Quemada write that Cruveilhier's description remained "unattended for two decades," in Berrios and Quemada, 178.

28. 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. Charcot pointed out that, "un grand intérêt s'attache à la histoire de cette gangue conjonctive, principalement pour le pathologiste; car c'est à elle qu'il faut attribue le rôle capital dans certaines altérations des centres nerveux et en particulier dans les cas qui nous occupent. He then cited two previous works: "on sait que les premières études sur la gangue conjonctive de la moelle épinère remontent à 1810 et sont dues à Keuffel; mais ce que l'on sait moins, c'est que Cruveilhier dans son article Apoplexie, du dictionnaire de médecine et de chirurgie pratiques, publié en 1820, a mentionneé, 'le tissue cellulaire séreux extrémement délié qui unit et sépare les fibres cérebrales et qui forme une trame excessivement tenue,'" Charcot, ibid., 554.

29. Charcot did not even cite Frerichs of Breslau's 1849 study.

30. Clymer, 229.

31. Démosthène, 12.

32. Timme, 1922, 5.

33. Clymer, 229. Goetz, Bonduelle, and Gelfand, 113.

34. Charcot, "Sclerosis in Scattered Patches," in *Leçons sur les Maladies du Système Nerveux faites à la Salpêtrière, Première edition*, 168-219, trans. Oliver, 52. Démosthène wrote that, "en 1862, Skoda, et en 1864, Zenker en Allemagne, publient quelques observations sous le titre de paralysie agitant, parmi lesquelles la sclérose en plaques est confondue," Demosthène, 13. <In 1862, Skoda and in 1864, Zenker in Germany, published some observations under the title paralysis agitans, among those which multiple sclerosis is confused.> Translation mine.

35. Rosenberg, "Introduction," *Framing Disease*, xiii. Of course, the afflicted did experience distress regardless of the naming of their malady.

36. "During their early years at the Salpêtrière, Charcot and Vulpian collaborated so closely in the study of multiple sclerosis and Parkinson's disease that the exact contribution made by each cannot be determined," Goetz, Bonduelle, and Gelfand, 113.

37. Charcot, "Histologie de la sclérose en plaques," *La Lancette Française Gazette Des Hopitaux Civils et Militaires* 41, no. 140 (1 Dec 1868): 554.

38. "Quelques années plus tard, de 1862-63, Charcot et Vulpian donnent à la

maladie le nom de sclérose en plaques disséminées; désormai la maladie porte une étiquette qui la distingue; elle est reconnue comme indvidualité morbide," Démosthène, 12. Goetz, Bonduelle, and Gelfand, 113. Charcot, "Sclerosis in Scattered Patches," trans. Oliver, 721.

39. Ibid., 721.

40. Ibid., 722.

41. Ibid., 724.

42. "Il sera, je crois, avantageux d'inaugurer cette étude par l'examen de tranches minces, transparentes, pratiquées transversalement sur des troncons de moelle convenablement durcis dans une solution d'acide chromique, et colorés par le carmin. Le carmin est ici un réactif précieux. Grâce à lui, certains éléments qui ont la propriété de se colorer sous son influence d'une teinte plus ou moins vive, sont par là mis en relief, alors que les autres conservent leur aspect ordinaire. Ainsi les cellules ganglionnaires, leur noyau, leur nucléole et aussi les prolongements de ces cellules, se colorent fortement sous l'influence de ce réactif. La gangue conjunctive se colore également dans tous les points de son étendue, à la vérité d'une manière bien moins prononcée: et, pour ce qui a trait aux tubes nerveux, seul le cylindre d'axe prend la couleur du carmin, tandis que l'enveloppe de myéline résiste complétement à son action," Charcot, "Histologie de la sclérose en plaques," 554.

43. Charcot, *Histologie,* parts two and three. McAlpine, *Multiple Sclerosis*, 2.

44. Charcot, "Sclerosis in Scattered Patches," trans. Oliver, 1014.

45. Ibid., 52-54. Charcot emphasized that "the trembling of which we speak does not show itself except on the occasion of intentional movements of a certain extent; it ceases to exist when the muscles are left to complete repose. Such is the phenomenon which I have been led to consider as one of the most important clinical characters of cerebro-spinal sclerosis in patches," ibid., 53.

46. Ibid., 54.

47. Ibid., 54-55.

48. Ibid., 55.

49. Ibid., 117-18.

50. Ibid., 119-120.

51. Ibid., 120-24.

52. Clymer, 230. Goetz, Bonduelle, and Gelfand slice the last sentence of this quote when they attribute the discovery of multiple sclerosis to Charcot, see Goetz, et al., 119.

53. J. M. Charcot, *Lectures on the Diseases of the Nervous System, second series*, trans. and ed. George Sigerson (New York: Hafner Publishing Company, 1962), 7. For the use of inflammation as a generalized pathological category in the nineteenth century see John Harley Warner, *The Therapeutic Perspective: Medical Practice, Knowledge, and Identity in America, 1820-1885* (Cambridge, Mass.: Harvard University Press, 1986), 68.

54. Clymer, 226.

55. Ibid., 231-33.

56. Richard Lechtenberg, *Multiple Sclerosis Fact Book* (Philadelphia: F.A. Davis Company, 1995), 10.

57. Fredrikson and Kam-Hansen, 237-43. Goetz, Bonduelle, and Gelfand write that "Charcot's mentor in the field of multiple sclerosis was the famed Cruveilhier, who had originally described "island of sclerosis" in his *Atlas of Pathologic Anatomy* (1829-1842)," in Goetz, Bonduelle, and Gelfand, 116. This statement is wrong because the one particular drawing of Cruveilhier in question, from 1838, was forgotten for twenty years and Charcot did not cite him as his mentor in the field of multiple sclerosis.

58. Charcot, "Sclerosis in Scattered Patches," trans. Oliver, 50-55. Clymer, 226. Writing in 1873 C.H. Boardman pointed out that myelitis conveniently accounted "for various diseases of the nervous system with which we know now <1873> it has little if anything in common," C.H. Boardman, "Progressive Multiple Cerebro-Spinal Sclerosis," *The Northwestern Medical and Surgical Journal* 3 (1873): 256.

59. Foucault, *The Birth of the Clinic*. Maulitz, *Morbid Appearances*. Warner, "Remembering Paris." Risse, "A Shift in Medical Epistemology," 115.

60. For the relationship between new institutions and new medical technology in the twentieth-century see, Joel Howell, *Technology in the Hospital: Transforming Patient Care in the Early Twentieth Century* (Baltimore: Johns Hopkins University Press, 1995), 1-20.

61. Berrios and Quemada, 176.

62. "Charcot's longitudinal assessments, from the early phases of illness through severe disability, permitted a detailed clinical description of the evolution of the various signs seen in multiple sclerosis. Furthermore, his access to large numbers of chronically disabled patients allowed him to note the youthful age of onset and female predominance in contrast to the older onset and male predominance of Parkinson's disease," Goetz, Bonduelle, and Gelfand, 116. Goetz, Bonduelle, and Gelfand perhaps overstate the importance of Charcot's correlation of gender with multiple sclerosis because, as Anthanassio Démosthène noticed in 1872, "d"après M. Charcot, la maladie serait plus commune chez les femmes: sur un total de 27 cas, il a trouvé 5 hommes et 22 femmes, mais cette proportion ne saurait être définitivement exacte, parce que, comme le font remarquer MM. Bourneville et Guérard, plusieurs médecins qui se sont occupé de cette maladie étaient attachés à des établissements réservés aux femmes," Démosthène, 15 . Translation mine: "according to M. Charcot, the disease might be more common in women; of a total of 27 cases, he found 5 men and 22 women, but this proportion might not be definitively exact, because as mentioned by MM. Bourneville and Guérard, many physicians who occupied themselves with this disease were attached to establishments reserved to women."

63. "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 autre 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, "Histologie de la sclérose en plaques," 557.

64. Goetz, Bonduelle, and Gelfand, 8-9, 72-74.

65. Charcot's long tenure at a single institution, the Salpêtrière was unusual. Joffroy and Bourneville, two of Charcot's students, recognized the importance of Charcot's "long-term link" to the institution in fostering his contributions. See Goetz, Bonduelle, and Gelfand, 38. After Charcot returned to the Salpêtrière in 1862 he, together with Vulpian, inventoried the enormous patient population. "For the first time in the two-century existence of the institution, medical case histories were compiled on its vast population; these patients could now be followed prospectively and, when the occasion arose, the clinical record would, as at the general hospitals, be correlated with autopsy findings using modern methods of gross and microscopic analysis. From this source, Charcot and Vulpian derived numerous joint publications during the 1860s," ibid., 39.

66. Goetz, Bonduelle, and Gelfand, 39.

67. Ibid., 69. "Although this work occupied Charcot throughout his professional

career, it became highly developed in the years between 1862 and 1875, and the large axes of this effort were constructed in this determinant period of his life," ibid., 66.

68. "The Germanic laboratory model was categorically different from the French hospital research system, in that the German laboratories and histopathological institutes operated outside the hospitals and were not specifically allied to clinical correlation," ibid., 73.

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

70. Ibid., 52.

71. Ibid., 1034-1035.

72. Ibid., 633-634.

73. Guenter B. Risse, "The Anatomical-Clinical Synthesis: From Morgagni to Laennec," in *Histoire de la PensÈe MÈdicale Occidentale*, ed.M. Grmek, 177-197 (Paris: Ed. Seuil, 1997).

74. Goetz, Bonduelle, and Gelfand, 116.

75. Ibid., 70-71.

76. Berrios and Quemada, 174. Roy Porter has found that Charcot "with the backing of his colleagues and students and with unlimited access to clinical material, mobilized a research industry," in Roy Porter, "Chapter 4 Parkinson's Disease (Paralysis Agitans) Social Section," in Berrios and Porter, *A History of Clinical Psychiatry*, 116.

77. On the newness of the histopathological technology, it must be remembered that "Virchow launched his cellular theory in 1858, a few years after Charcot completed his medical training. He <Virchow> demonstrated that tissues were composed of cells that maintained a normal order unless disrupted or destroyed by disease states. Virchow opened the era of the microscopic study of anatomy, surpassing the macroscopic studies of the French in novelty as well as depth and detail. The French first edition of Virchow's work on cellular pathology appeared in 1861 . . . ," Goetz, Bonduelle, Gelfand, 73. The seminal years in the construction of the disease category of multiple sclerosis were 1862 to 1872.

78. Ibid., 22-23.

79. To see the effect of translating European medicine into American contexts for the early and middle nineteenth century see Lisa Rosner, "Thistle on the Delaware: Edinburgh Medical Education and Philadelphia Practice, 1800-1825," *Social History of Medicine* 5 (1992): 19-42. See also, Warner, "Remembering Paris," 301-325.

80. Robley Dunglison, *A Dictionary of Medical Science* (Philadelphia: Blanchard and Lea, 1860). J. Thomas, *A Comprehensive Medical Dictionary* (Philadelphia: J.B. Lippincott & Co., 1865). Robley Dunglison, *A Dictionary of Medical Science* (Philadelphia: Henry C. Lea, 1868), 870.

81. C. Handfield Jones, *Clinical Observations on Functional Nervous Disorders, Second American Edition* (Philadelphia: Henry C. Lea, 1868). The author was British but the textbook was printed for American audience.

82. J.C. Morris, "Case of the late Dr. C.W. Pennock," *American Journal of the Medical Sciences* 111 (July 1868): 138-144.

83. Doctors diagnosed Pennock with phlegmasia dolens during life. S. Weir Mitchell, with regard to the microscopical examination wrote: "as regards the condition of the spinal cord, nothing can be more striking than the confirmation it gives of the views of Brown-Séquard as to the mode of transmission of voluntary motor power throughout the antero-lateral and anterior columns. If we suppose that the long-continued over-exertion of his physical powers had caused a destruction or atrophy of a portion of the motor cells of the anterior cornua, followed by atrophic retrogressive gelatiniform degeneration of the

nerve tubules proceeding from these cells, and that this process was repeated as the remaining cells were brought into excessive action to maintain the functions of life, a result more closely resembling that observed in this cases would be obtained than that from any other hypothesis I can form," ibid., 143-44.

84. M. Gonzalez Echeverria, "Sclerosis of Both Third Anterior Frontal Convolutions Without Aphasia," *The Medical Record* 4 (1869): 1-2.

85. Ibid., 1-2.

86. It seems that this ahistorical appropriation of past work is a constitutive cultural practice of modern medicine.

87. Clymer, 227.

88. Ibid., 231. Clymer articulated a refinement of Virchow's definition of sclerosis: Clymer wrote: "Virchow says, 'Sclerosis . . . signifies thickening with condensation.' this definition is too limited and vague. The term is strictly descriptive, not of induration from any cause, but of changes in the textural condition of an organ. It involves both quantity and quality. There is overgrowth (proliferation) and transformation of connective tissue, with consequent wasting of the proper functional elements of the part . . . so sclerosis of the nervous centres means not only parasitic exuberance of the connective gangue (neuroglia), but proportional compression, deterioration, and annihilation of the ganglion-cells and nerve-tubes. There is histological substitution . . . Comprising what has hitherto been treated of by writers under the name of chronic inflammation of the brain and spinal cord--chronic encephalitis and chronic myelitis,-it is only lately that its true pathogeny has been intelligently set forth. Morbid conditions, hitherto confounded but essentially distinct, have been differentiated, and the proper signs of each modality established," ibid., 226.

89. Ibid., 246-47.

90. Henry D. Noyes, "A Case of Supposed Disseminated Sclerosis of the Brain and Spinal Cord," *Archives of Scientific and Practical Medicine* 1 (1873): 43-46.

91. Boardman, 251.

92. J. K. Bauduy, "Multiple Cerebro-spinal Sclerosis," *Missouri Clinical Record* 1 (1874): 4.

93. George S. Gerhard, "Cases of Multilocular Cerebro-Spinal Sclerosis," *Philadelphia Medical Times* 7 (November 11, 1876): 50

94. William Alexander Hammond, *A Treatise on Diseases of the Nervous System* , first edition (New York: D. Appleton and Company, 1871), second edition 1872. For background on Hammond see, Bonnie Ellen Blustein, *Preserve Your Love for Science: Life of William A. Hammond, American Neurologist* (New York: Cambridge University Press, 1991).

95. Hammond, *A Treatise on Diseases of the Nervous System*, 637 .

96. Ibid., 288.

97. Ibid., 288.

98. Ibid., 291.

99. John I. Cook, "Multiple Cerebro-Spinal Sclerosis," *The Richmond and Louisville Medical Journal* 14 (1872): 76-78. Hammond had reinterpreted old cases.

100. Stiles Kennedy, "Cerebro-Spinal Sclerosis, Involving the Hemispheres," *Detroit Review of Medicine and Pharmacy* 8 (1873): 99-102.

101. Horatio C. Wood, Jr. "The Multiple Scleroses," *The Medical Record* (9/14/1878): 224-25.

102. Hamilton, 346. Regarding the fluid boundaries between chorea and multiple sclerosis into the 1910s, Charles Davenport reported that during the World War I mobilization in the United States "it is, of course, possible that in the rapid diagnosis of local boards and camps some cases of choreas may have been diagnosed as multiple

sclerosis and vice versa," Charles Davenport, "Multiple Sclerosis from the Standpoint of Geographic Distribution and Race," in Association for Research in Nervous and Mental Disease, *Multiple Sclerosis [Disseminated Sclerosis], volume II* (New York: Paul B. Hoeber, 1922), 10.

103. Hamilton, 348-49.

104. M. Rosenthal, *A Clinical Treatise on the Diseases of the Nervous System, with a preface by professor Charcot*, translated from the author's revised and enlarged edition by L. Putzel, M.D. (New York: William Wood & Company, 1879), 87.

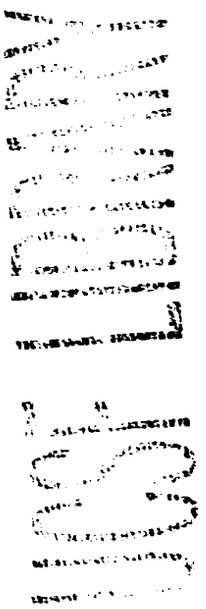
105. Joseph O. Hirschfelder, " Disseminated Sclerosis: A Clinical Lecture Delivered at the Medical College of the Pacific,' *Pacific Medical and Surgical Journal* 25 (1882): 433-449 (some pages misnumbered). See also Arch. Dixon, "A Case of Disseminated Sclerosis," *Alienist and Neurologist* 3 (1882): 50-57. Dixon cites Charcot directly as well.

106. Sherwin, 39.

Chapter Two
The Rise of Multiple Sclerosis as a Disease Problem in The United States, 1870-1960

I Introduction

Investigating how and why diseases such as leprosy, plague, smallpox, syphilis, yellow fever, malaria, tuberculosis, rheumatic fever, and AIDS rise and fall has been a fundamental area of inquiry in the historiography of disease.[1] Other scholars interested in the waxing and waning of diseases have included: social historians concerned with the causes of the health revolution of the late nineteenth and early twentieth centuries; demographers interested in broad trends of mortality, morbidity, and population rise; and the new materialists who use an ecological approach to explain, for example, the Columbian Exchange.[2] Other historians have examined the ways in which interest in diseases can be intensified because of larger social stresses such as political struggles, war, and mass immigration.[3] In addition, cultural historians have analyzed the rise, fall, and mutation of various disease categories as social constructions such as: neurasthenia, chlorosis, homosexuality, drug addiction and alcoholism, masturbation, anorexia nervosa, hysteria, and chronic fatigue syndrome.[4] These cultural analysts have tended to emphasize questions of social control and conflict, deviance, gender, and professional power. Their focus on conditions where an underlying pathophysiology was problematic has served to highlight the socially constructed nature of disease categories.



This attention to the historical contingency of nosological categories has problematized simplistic explanations of the rise and fall of diseases. For example, Andrew Cunningham has argued that there was a radical discontinuity between the identity of the new plague and that of the old plague. After 1894 the plague's identity depended exclusively on laboratory proof of the presence of *yersinia pestis*. Before the rise of the laboratory, physicians constructed plague's identity through reference to symptomatology.

Thus, the boundaries of what constituted "plague" were much more fluid before 1894. Therefore, for Cunningham, historians cannot reliably assume that "plague" in the past was the same as "plague" in the present.[5]

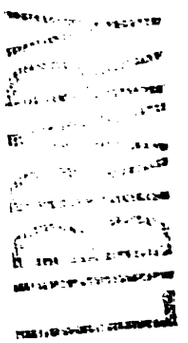
Even in the 1940s in the United States plotting the rise and fall of a disease as well-known as malaria proved to be difficult as Margaret Humphreys has shown. Many southerners saw fevers and chills as such common events that they did not report the cases to doctors and sometimes did not even consider them illnesses. "Southern physicians were prone to call all fever, malaise, myalgias, and headaches 'malaria' and to treat these symptoms with quinine."[6] When the Centers for Disease Control investigated blood smears from 1,162 "malaria" cases from Alabama in 1947, they confirmed none as malarial.[7]

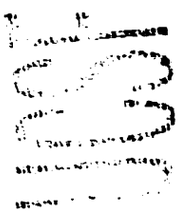
As Guenter B. Risse has pointed out, in addition to the fluid boundaries of past nosologies one faces the problem of the shifting ecology of disease; this means that the incidence, patterns, and combinations of material conditions that a culture might frame as a "disease" change over time in a specific place. One example is the mid-twentieth-century shift from an ecology of disease dominated by infectious conditions to one dominated by chronic maladies and cancer.[8]

In addition to shifting nosologies and shifting material conditions, the death and morbidity records historians often use are problematic sources. What constitutes a cause of death itself is dependent on signs, markers, symptoms, and epistemologies that shift in time and place. The records are places of contest, subterfuge, obfuscation, negligence, and choice where who died, of what, according to whom, as recorded by whom, according to multitudinous cultural, religious, political, and economic reasons further distorts the information. This makes the precise ciphering of the causes of, for example, the great mortality decline, a difficult methodological problem because of the dynamic and interactive

character of multiple categories the historian of disease must analyze.[9]

One way to solve the problem is to combine the ecological, cultural, and social approaches so that they might inform one another. Robert A. Aronowitz has defined disease ecology to mean "the interdependence among prominent diseases at any particular time and place."[10] Shifting ecologies of disease can affect the perception of physicians about the nature of disease in general by presenting different material disease problems to their collective consciousness.[11] However, differing ecological conditions can only partly explain the clinical framing of symptoms. For example, that chronic fatigue syndrome received increased attention was, according to Aronowitz, only partly because of the heightened interest in immune system disease in general caused by the AIDS epidemic. The reason for the increased attention was not because of "biological similarities" but because of "related controversies" such as the disease attacking specific social groups, the marginalization of patients, and the stigmatization of patients by the medical establishment and lay public.[12] In other words, shifting cultural and social contexts can impact medical interpretation of embodied clinical pathology.





The rise of multiple sclerosis in the United States shows how a shift from one disease ecology to another can change clinicians' perceptions of embodied symptoms and it demonstrates how attention to the historical contingency of disease categories and the historical contingency of health practices which generate nosologies contributes to and complicates explanations concerning the waxing and waning of diseases in the past.

II The Historical Problem

Neurologists considered MS a rare disease in the United States in the late nineteenth century.[13] For example, from 1883 to 1906 New York Hospital physicians diagnosed MS ten times for in-patients. They diagnosed MS five times at the out-patient House of Relief

from 1892 to 1906.[14] In 1892 Charles Dana reflected the standard view among neurologists when he wrote that "in America the disease is, in the writer's experience, rare."[15]

In the early twentieth century some neurologists began to question this finding because they recognized how difficult it was to diagnose the condition and comparative figures from Europe cast doubt on the apparent paucity of MS cases in America.[16] Nevertheless, the general perception remained through the 1910s that MS was rare in the United States.[17]

These statistics and continuing suspicions about misdiagnosed cases led, during most of the 1920s, to assertions by neurological authors that MS was an infrequent disease but one that was increasing slowly.[18] However, the actual diagnosis of MS in the clinic remained uncommon. In 1928 two physicians reported that ". . . at the Philadelphia General Hospital from 1920 to 1926 inclusive there were studied 6,974 cases in the neurological wards and the diagnosis of multiple sclerosis was made twenty-four times (0.33 percent) . . ."[19]

By the early 1930s many neurologists' perception of the frequency of MS had begun to change.[20] In 1931 Sidney D. Wilgus and Egbert W. Felix wrote that "Multiple Sclerosis is a common disease, and hence the importance of recognizing its early symptoms is worth emphasizing."[21]

This shift in perception continued in the early and middle 1940s.[22] Dr. Tracy Putnam wrote in 1943 that "it is clear that in this locality <New York City> at least, multiple sclerosis is by no means a rare disease."[23] By the late 1940s and early 1950s some American neurologists saw MS as perhaps the *most* common disease of the central nervous system in the United States. In 1948 one group of neurological authors estimated that there were 50,000 to 90,000 MS patients in the United States.[24] In that same year

Charles C. Limburg estimated that there might be as many as 150,000 MS cases.[25] In 1949 Wisconsin Professor Hans Reese declared that "among the diseases of the central nervous system, multiple sclerosis ranks today almost as the most frequent illness."[26] In 1954 O.E. Buckley estimated that there were 200,000 to 300,000 cases of MS in the United States.[27] In 1960 Vermont professor George A. Schumacher agreed that there were approximately 250,000 multiple sclerotics in the United States.[28]

What explains this transformation of the perception of the frequency of MS in the United States? Was it due to an actual increase in the underlying pathology of demyelination and overgrowth of glial tissue in the population, or an increasing knowledge of the disease, or earlier diagnoses of the condition, or a complex interaction of social, professional, cultural, and ecological processes?

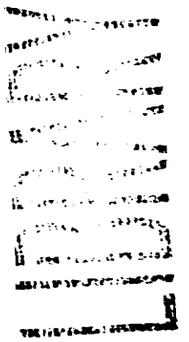
III American Professional and Social Structures

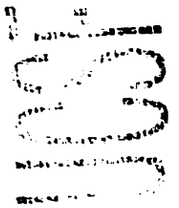
One reason neurologists saw more cases of MS gradually from the 1870s to the 1950s was because American neurology's ongoing process of specialization during this period made diagnoses of MS increasingly possible. Elements of this process included: improved training of neurologists, greater experience, greater numbers of neurologists, a decrease in competition with alternative healers, greater access to patients and autopsies, the increasing urbanization of the United States, and more stable population groups on whom to practice.

American neurology's professional structures and boundaries evolved slowly from 1872 to 1934 as did the structures of most middle-class professions in the United States.[29] American medical schools did not teach neurology as a specialty in the United States before the Civil War, the first professorships emerging in the 1870s. Physicians concerned with neurology founded the first specifically neurological association in America in 1872 and

self-defined neurologists formed the American Neurological Association in 1875.[30] In the 1870s one sees incipient specialty formation and not a mature research community.[31]

Besides the "chaos" within American neurology, it existed in a highly competitive and little regulated medical marketplace in the late nineteenth century.[32] The boundaries between neurologists, alienists, psychiatrists, general practitioners, neurosurgeons, gynecologists, osteopaths, faith healers, christian scientists, and hypnotists remained blurry in the United States well into the twentieth century.[33] These various health practitioners sought out nervous patients and their fees. Likewise, American patients eagerly embraced alternative health workers. This meant that neurologists were less likely to encounter large numbers of nervous patients who simply went elsewhere to practitioners with little or no knowledge of the then obscure disorder. The numbers of alternative practitioners declined markedly from 1900 to 1920 as a result of vigorous state intervention and a maturation of the professionalization process in American medicine.[34]





While medicine in general had secured professional boundaries by the 1920s, the dynamic process of medical specialization continued in the 1920s, 30s, and 40s.[35] This process of specialization could impact the kind of diagnosis a physician might give a patient. For example, warring neurologists and psychiatrists were not able to clearly demarcate professional boundaries with respect to the rest of the profession and one another until the founding of the American Board of Psychiatry and Neurology in 1934.[36] In practice, the American Board of Psychiatry and Neurology certified most diplomates in psychiatry and neurology; however, psychiatrists had far more prestige than neurologists in the 1930s in the United States. This meant that intellectually there were clearer boundaries but in practice there were not. In terms of the other closest professional boundary, a small number of students chose certification in neurology and neurosurgery. This did not become problematic in terms of professional politics until the 1940s.[37]

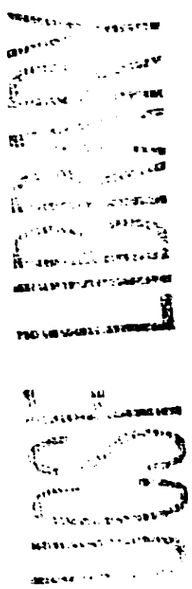
The codification and bureaucratization of neurology in the 1930s *coincided* with the changed perception of MS as a common disease in the 1930s. Commenting on the importance of professional structures in seeing multiple sclerosis, Maurice Fremont-Smith wrote in 1929 that "it is not to the neurologist but to the internist and surgeon that these <MS> cases should be of interest. Two of these patients had been repeatedly examined by internists of ability; one had been in the hands, successively, of two of our best orthopedic surgeons. That the diagnosis was suspected by no one of these men (as it was not), shows the total lack of familiarity with this disease that exists outside the neurological group. You will recall that one case had a needless major operation and was about to be submitted to a second."[38] As there were more neurologists, and as their specialty's boundaries and authority became more secure, there were more cases of MS.

The urbanization of America during this period also contributed to the increasing numbers of MS cases reported by concentrating a greater percentage of the American population and thus patients within the analytic view of the mostly urban neurologists.[39] Academic neurologists had known of the disease since the 1870s; however, from the 1870s to the 1920s the advanced neurological and clinical training and experience necessary to confidently diagnose MS existed in only a few cities, notably New York City and Philadelphia and to a lesser extent Boston, Chicago, Baltimore, Washington, and St. Louis.[40] It is no surprise that most of the journal literature on MS from the 1870s to the 1920s comes from Professors in New York City and Philadelphia. However, most Americans and thus patients lived in rural areas and small to medium sized towns before the 1920s and thus did not usually have access to the highly trained neurologists of the big cities; therefore, diagnoses of MS were less likely under these structural conditions.[41]

Where most neurologists did practice from the 1880s to 1910s, mobile immigrants filled the cities.[42] It is not clear how population mobility might have impacted long-term

follow-up in terms of private practice. However, the cost of an expensive consultation would have been prohibitive to a great number of nervous patients. In terms of the urban charity hospitals and out-patient clinics where many neurologists consulted, high patient mobility did reduce the number of MS diagnoses given. As S.G. Webber of Boston noted in 1905: "this difficulty of diagnosis, especially in the earlier stages of the disease, may in part explain its apparent rarity. Patients attend a dispensary when the diagnosis is uncertain, hence the disease is not recognized. When more advanced they are not seen, because by that time their condition is considered hopeless and a physician is sent for only in some emergency, therefore their cases are never reported."[43]

The underdeveloped specialty structure of American neurology, the mostly rural and small-town practice of American medicine, and the highly mobile population on whom urban neurologists practiced partly ensured that MS would be a rare disease in the United States until after the 1920s. Patients with the underlying process of demyelination could have been out there but the training required to see it was not easily obtained and the structural contexts of medical practice made the diagnosis even less likely.



IV A Difficult Diagnosis

Another reason for the rarity of MS in the United States before the 1930s was that it was a very difficult disease to diagnose even for the most highly trained elite neurologists.[44] This was because, as William Hammond said in 1872, "few diseases are so irregular and ununiform in their phenomena as the cerebro-spinal form of sclerosis."[45] Hugo Engel pointed out in 1879 that: "only a few years ago a celebrated professor, when lecturing on paralysis agitans, included in his classical description of this disease many symptoms which we now know, at least since Charcot's clinical observations, are diagnostic of an entirely different disease, viz., multiple cerebro-spinal sclerosis."[46] San

Francisco professor Joseph Hirschfelder discussed the difficulties the differential diagnosis of multiple sclerosis could pose in a clinical lecture at the Medical College of the Pacific in 1882. The case he discussed had "produced the signs of tabes dorsalis" which could be easily confused with disseminated sclerosis. Hirschfelder then recounted "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."[47] Likewise, Theodore Diller of the St. Francis Hospital in Pittsburgh, Pennsylvania reminded his listeners at the Pittsburgh Academy of Medicine in 1895 that "nearly all authorities agree that insular sclerosis often presents great difficulties in the way of diagnosis."[48]

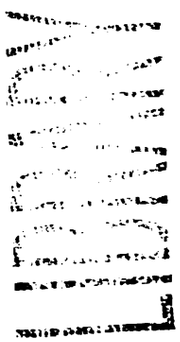
This difficulty partly resulted from the polyphonic symptoms which result from the pathophysiological process of demyelination and overgrowth of glial tissue in the brain and spinal cord. In 1898 Bernard Sachs listed the spinal and cerebral symptoms which might appear in MS: the spinal symptoms might include: intention tremor, titubation, contractures; the cerebral symptoms might include: dysarthria, scanning speech, nystagmus, vertigo, transitory amblyopia or diplopia, apoplectiform or epileptiform attacks, difficulty in deglutition, mental enfeeblement; other symptoms might include: scattered paresthesias, muscular atrophies, lightening pains, and gastric and bladder problems.[49] These symptoms could appear in various combinations and could remit for years. This obviously made diagnosis difficult.

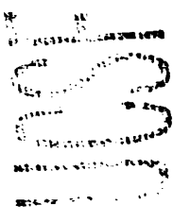
We can see in detail how the complicated nature of the diagnosis might vex even

elite neurologists in the following case from Charles W. Burr, neurologist to the Philadelphia Hospital, and D. J. McCarthy, Associate in Medicine in the William Pepper Clinical Laboratory of the University of Pennsylvania. The doctors examined Mr. D. in 1899. Seven years previously the patient "began to have difficulty in walking" and by 1899 could not "move his legs at all." He had at times a "slight spasmodic retention of urine" and "during the past year his bowels" had "been moved only after the use of purgatives. The necropsy revealed <MS> . . . He died suddenly a few weeks after our examination . . ." During Mr. D.'s lifetime physicians had diagnosed him with posterior sclerosis, an ataxic paraplegia (postero-lateral sclerosis), and spastic paraplegia. The physicians maintained that during his lifetime "the typical symptoms of multiple sclerosis" were "never at any time" present. This was because of "the course of the disease itself, which, by picking out certain system tracts of the cord at successive intervals, led to the diagnosis by different observers of the disease suggested by the tracts affected."[50]

The complicated nature of the MS diagnosis remained a problem for many neurologist through the 1920s. At a 1921 neurological conference devoted to MS, in a discussion following a paper by Bernard Sachs and Dr. Emanuel D. Friedman, Dr. <Ramsey> Hunt asked Dr. Sachs: "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 saying that " 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 . . . 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 at this point at least the first 9 groups of symptoms and try to make the diagnosis, if any number of these are present."[51]

The diagnosis of MS remained difficult from the 1920s through the 1950s. New laboratory technologies such as the Wasserman test, the colloidal gold test and cell counts of the spinal fluid did not make the diagnosis of MS easier. (The Wasserman test will be dealt with in more detail in the next section on differential diagnosis). A 1948 discussion at a conference devoted to MS by the Association for Research in Nervous and Mental Diseases shows the limited usefulness of the laboratory in diagnosis. David A. Freedman and H. Houston Merritt had presented their study on the cerebrospinal fluid of MS patients.[52] They noted that "the effort to establish a relation between the various abnormalities noted and other aspects of the disease process has been a constant, if not a very rewarding, one."[53] Though some neurologists were promoting the colloidal gold curve as a promising diagnostic test, in practice it was unreliable. This was because "the difference in incidence of colloidal gold curves obtained" varied depending on the "difference in technic" of individual laboratories. Tracy Putnam reported that at the City Hospital in Boston they had "a very high incidence of abnormal colloidal curves in cases of multiple sclerosis. If I remember rightly it was something like 60 or 70 per cent." However at the New York "Neurological Institute . . . the incidence was very much lower. My guess is that it was around 10 or 20 per cent."[54]





Dr. Theodore J. von Storch of Albany, New York clarified why, for example, a positive colloidal gold curve in the presence of a negative Wasserman was not pathognomonic: "we feel our observations parallel those of Dr. Freedman and Dr. Merritt. We also feel that neither the gamma nor the Type D curve is specific for multiple sclerosis. We have found that Type D curve in other disorders, even as an increase in gamma globulin. We should like to emphasize that neither test is specific, and the test is considered by Dr. Lange to be highly suggestive but certainly not diagnostic, and certainly not even characteristic."[55] Dr. H. Houston Merritt of New York City concurred that there

were many problems with the tests. He also agreed that "there is no specific finding in the cerebrospinal fluid that will make the diagnosis of multiple sclerosis."[56]

Thus, the keys to diagnosis remained clinical. In 1960, Vermont Professor George Schumacher taught what was necessary to diagnose MS: that is, unequivocal evidence of scattered lesions throughout the central nervous system and for many patients evidence of remissions. With regard to laboratory technology Schumacher wrote that "no specific diagnostic test for multiple sclerosis is available. None of the abnormal laboratory results found in multiple sclerosis is present in all cases and none is pathognomonic of the specific lesion of the disease . . . Pneumoencephalograms show symmetric or asymmetric cerebral atrophy of slight to moderate degree in moderately advanced cases, but this nonspecific finding does not warrant the use of the procedure as a routine diagnostic test . . . Examination of the cerebrospinal fluid is sometimes of value in the diagnosis of multiple sclerosis and should be carried out routinely." However, with regard to the colloidal gold test he held that "the reaction . . . cannot be considered pathognomonic." As for the new work on protein fractions "no deviation has been claimed to be pathognomonic."[57] Schumacher concluded that "numerous biochemical, hematologic and physiologic abnormalities that occur in most patients with multiple sclerosis are either too nonspecific for use as diagnostic features or involve technics of measurement more suitable for the research laboratory than for the clinical hospital laboratory. . . . These alterations have not yet served to provide help in diagnosis or understanding of pathogenesis."[58] Diagnosis was made "most dependably on the basis of history and neurologic findings."[59]

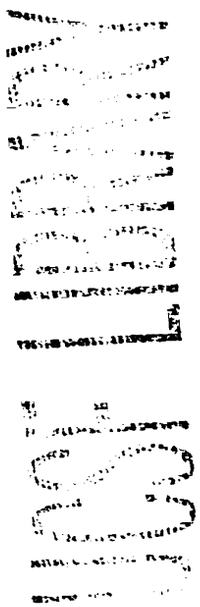
In my reading of 313 patient records from Tracy Putnam's private neurological practice in Beverly Hills and the University of California, Los Angeles Hospital and Neurology Clinic <UCLA> records, from 1947 to the early 1960s, I do not recall one case where the laboratory results overturned the clinical diagnosis of MS.[60] Thus, the clinic still

came first and the new laboratory technologies did not significantly increase the number of MS cases seen.

The protean biology and lack of pathognomonic laboratory signs of MS created the possibility for wide diagnostic interpretive possibilities. Physicians did not uncover more cases of MS so much as they reinterpreted and reinscribed patients bodies who presented certain symptoms.

V The Emergence of MS from other Disease Categories

Physicians in the 1920s, 30s, and 40s remarked that they tended to recognize MS earlier in the disease course than previous generations of doctors had.[61] This was accurate but it was not that earlier physicians saw nothing when they encountered patients with the underlying pathophysiology of demyelination; it was that they interpreted the symptoms differently. Examining several of these diseases, or nosological neighbors, in more detail explains where most of the rise in MS cases came from: that is, they emerged out of other disease categories, the most important of which were syphilis of the central nervous system and hysteria. This process continued through the 1950s.



The first major nosological category out of which MS emerged was paralysis agitans. In 1870, Meredith Clymer remarked that "all the English authors confound this disorder <MS> with paralysis agitans. Parkinson, whose description of shaking palsy has been closely followed unquestionably did . . . Dr. W. R. Saunders . . . in an excellent article on paralysis agitans, confuses it with diffuse cerebro-spinal sclerosis."[62] In 1876 Jerome K. Bauduy, instructed that "until within comparatively a very recent period paralysis agitans, multiple cerebral sclerosis and multiple cerebro-spinal sclerosis were inextricably confounded together under the one common name of paralysis agitans.[63] In 1878 Allan McLane Hamilton, Attending Physician at the Epileptic and Paralytic Hospital,

Blackwell's Island, New York City and member of the American Neurological Association, wrote that "for a long time this disease was mistaken for paralysis agitans (Parkinson's disease), chorea, and other neuroses; and even after it had been shown to be a separate neurosis a certain amount of confusion existed in regard to its nomenclature and its position among the scleroses."[64] In 1879 Hugo Engel, Lecturer on Electro-Therapeutics at Jefferson Medical College and Physician to St. Mary's Hospital, remarked that "only a few years ago a celebrated professor, when lecturing on paralysis agitans, included in his classical description of this disease many symptoms which we now know, at least since Charcot's clinical observations, are diagnostic of an entirely different disease, viz., multiple cerebro-spinal sclerosis."[65] Also in 1879 A.B. Arnold, Professor of Diseases of the Nervous System, College of Physicians and Surgeons, Baltimore, Maryland wrote that " . . . the disease bears a close resemblance to shaking palsy with which it was formerly confounded, though the differential diagnosis offers no difficulties. Paralysis agitans, as a rule, is a malady of advanced age; the shaking is constant, whether the patient is at rest or intends to make a movement . . . "[66] In MS the tremor occurred with intentional movement only.

By the 1890s Eastern neurological professors, at least, felt confident in their abilities to discriminate between paralysis agitans and multiple sclerosis.[67] B. Onuf, Neurologist to St. Catherine's Hospital and Consulting Neurologist to the Hebrew Dispensary, in a paper read before the Kings County Medical Society on September 6, 1902 confidently asserted that "paralysis agitans is mentioned as a disease from which multiple sclerosis is to be differentiated, but I, personally, have seen very few cases in which the distinction was difficult, the ensemble of clinical picture differing on the whole so strongly in these two diseases that one could not long remain in doubt as to which of the two conditions was present."[68]

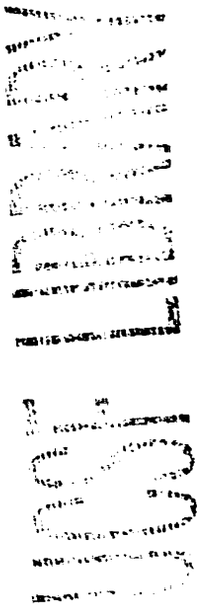
A clinical picture with symptoms such as a spastic gait, dysarthria, or tremor could also lead to a diagnosis of alcoholism from the 1870s to the 1950s. Meredith Clymer using a common trope in reporting a case from 1870 described a patient's "gait, which from the outset may have been more or less unsteady, is now staggering like that of a drunken man."[69] Similarly, another physician emphasized that for some patients "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."[70] In 1899 Memphis physician William Krauss related a case "of a white male, aged 28 years, laborer, who came under my charge with nausea, vomiting, great prostration, vertigo, anorexia, and constipation. He admitted having been on a spree, and, as he had some tremor, it was ascribed to that cause. A history of syphilis was denied; the family history was not inquired into. The diagnosis of alcoholism was entered." After giving strychnine, digitalis, arsenic (as general tonic) by day and sulfonal and morphine by night." Krauss recalled that "I began to suspect that there was a hole in my diagnosis. As I came upon him unawares I failed to notice any tremor, which, however developed after I spoke to him. I now felt certain that he was suffering from a progressive trouble, and a more careful examination and inquiry into his previous history was made . . . He had trembling for six months, which he ascribed to drinking, but it was getting worse, and he drank whiskey to 'steady his nerves.' All the reflexes were exaggerated, especially those of the knee and ankle; there were no areas of anesthesia." The man also had an intention tremor, nystagmus, and difficulty with speech. "The diagnosis of multiple sclerosis was made with some reservation, knowing the difficulty of correctly recognizing this condition. The differentiation lay between it and alcoholic tremor, results of slight encephalitis (red softening) or leptomeningitis. Chorea, paralysis agitans, hysterical and drug tremor were excluded." The patient did not have: girdle pains, optic symptoms, marked sensory disturbance, spastic paralysis, muscular atrophies and vertigo all which were

pathognomonic of MS but he did have Charcot's triad. "After a residence of five weeks in the hospital without improvement, he was sent to the county poor and insane asylum and was thus lost sight of."[71]

Spinal cord diseases also shared fluid nosological boundaries with MS from the 1870s to the 1950s. These included: syringomyelia, spinal tumors, dorsolateral sclerosis due to primary anemia <combined system disease>, transverse myelitis, chronic myelitis, spastic paraplegia, and ataxic paraplegia.[72] As I. Abrahamson admitted in 1902: "it was possible that we make mistakes in diagnosis in some cases of so-called acute or subacute transverse myelitis coming under observation as chronic transverse myelitis. Some of these cases would probably ultimately prove to be examples of multiple sclerosis . . ."[73] Bernard Sachs, remembering in 1917, said: "I think that formerly cases were largely classed as chronic myelitis and spastic paraplegia."[74] In the 1920s, increasing numbers of necropsies served to change clinical diagnoses of myelitis to postmortem diagnoses of multiple sclerosis.[75] On the fluid nature of the boundary between myelitis and MS, Tracy Putnam wrote in 1937 that "I shall make no attempt to set up criteria of differential diagnosis from disseminated encephalomyelitis, diffuse sclerosis, and neuroptic myelitis. It appears probable that these diseases represent variations of the same fundamental disease process, and transitional forms between the various groups occur."[76]

The boundaries between myelitis and MS remained blurry in the 1950s in the clinic as this example from The UCLA Hospital from June 5, 1958 shows: an Assistant Resident in Medicine recorded that the patient's "gait is shuffling with weakness in dorsiflexion of right foot and weakness of flexion of right hip. Patient is unable to walk on the toes of her right foot. She is not able to step onto a chair with right leg. In supine position the patient is unable to overcome gravity in flexing the right hip when elevating the right foot for dorsiflexion. Pos. <itive> Babinski on right and reflexes on the left are somewhat

hyperactive. Light touch sense absent and stocking distribution below the left knee and in small patches in the right leg below knee. Temperature reception is absent below the left knee. Pin prick over the entire left leg creates a burning sensation which is not perceived in the right leg . . ." The patient "was seen in consultation by Dr. #107 who felt that the patient had a myelitis of undetermined cause involving primarily the lateral fiber bundles on the right side at about the T-12 L-1 level. The possibility of early multiple sclerosis must be considered but can not be established without more definitive evidence of multiple lesions in the central nervous system."[77]



The most important nosological category of spinal diseases from which MS migrated and shared porous boundaries from the 1860s to the 1940s was tabes dorsalis, also known as locomotor ataxia.[78] Neurologists knew that the sclerotic lesions of tabes dorsalis chiefly affected the posterior columns of the spinal cord in the 1860s and 1870s whereas MS lesions could affect the anterior, lateral, or posterior columns. Because of where the lesions were on the spinal cord, many patients presented symptoms common to both disorders. As C. H. Boardman said in 1873: "it <multiple sclerosis> may simulate locomotor ataxy."[79] This made the differential diagnosis difficult. The problem was how to coordinate the physiological markers or clinical signs with the underlying anatomical lesions. The symptoms might include: tingling sensations, numbness, easy fatigue, or an ataxic gait. In 1880 George S. Gerhard taught how to make the differential diagnosis between multiple sclerosis, paralysis agitans, and locomotor ataxia. For multiple sclerosis the pathognomonic sign was the intention tremor; in locomotor ataxia the gestures were wild and abrupt; in paralysis agitans the tremor was constant. Physicians continued to write in the 1880s and 1890s on the difficulties of diagnosticating between tabes and MS and they frequently diagnosed the cluster of symptoms which could possibly lead to a diagnosis of MS as tabes dorsalis.[80]

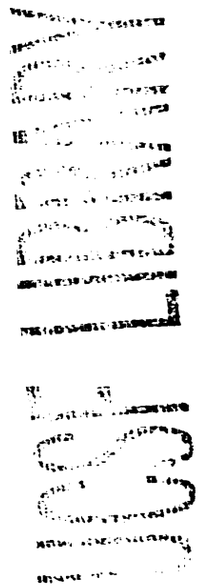
Syphilis of the central nervous system came in two other forms which often presented similar symptoms to MS: paresis and multiple cerebro-spinal syphilis. Bernard Sachs, in a discussion at the New York Neurological Society on December 1, 1898, discussed the problem of polyphonic and protean diseases: "there are several diseases of the central nervous system which are characterized by a muliplicity <sic> of lesions and a large variety of clinical symptoms. The most important of these affections are tuberculosis, cerebro-spinal syphilis, and multiple sclerosis . . . between multiple sclerosis and syphilitic diseases of the brain and cord there is the closest resemblance . . ."[81]

Sachs noted the problem of remissions common to both diseases which complicated the differential diagnosis and contributed to the two diseases' blurry nosological boundaries: "it is the occurrence of such remissions that makes it particularly difficult to distinguish between disseminated sclerosis and multiple cerebro-spinal syphilis . . . However clearly the symptoms may be developed, and however carefully, multiple sclerosis may be confounded with other diseases, above all with cerebral spinal syphilis . . .The remissions in the symptoms, the preponderance of the spasticity over the paralysis, the apoplectic seizures may be characteristic of both diseases."[82]

In 1902 B. Onuf concurred that ". . . cerebro-spinal syphilis . . . has many features in common with multiple sclerosis: First, the multiplicity of lesions. Second, the appearance in attacks separated by longer or shorter interals <sic> of relative freedom from new symptoms and of apparent stand-still of the disease. Third, the tendency of the symptoms to subside to a considerable extent . . . Spasticity is just as frequently observed in syphilis, the ataxy, at least of the lower extremities, is also quite frequent in syphilis, and the intentional tremor, on which so much diagnostic reliance is placed as in favor of multiple sclerosis, I have seen very typically developed in a case of undoubted cerebral syphilis."[83]

J. Ramsay Hunt, then instructor in neuropathology at the Cornell Medical School, reiterated one year later that "the diagnostic difficulties encountered in such cases are considerable. In the earlier stages of multiple sclerosis and dementia paralytica a certain resemblance is not infrequent, as many of the symptoms are common to both. Thus apoplectiform and epileptiform attacks occur frequently in both affections. Systemic degenerations in the posterior and lateral columns, so frequent in paresis, produce symptoms so similar to those following a development of plaques in the same areas."[84]

Differentiating between MS and syphilis remained difficult for neurologists in the first third of the twentieth century. It was even more problematic for the general practitioner who was much more likely to encounter a multiple sclerotic. However, new technology offered the possibility of making the differential diagnosis easier. Fritz Schaudinn and Eric Hoffman established the *Treponema pallidum* as the cause of syphilis in 1905.[85] In 1906, August Wasserman, Albert Neisser, and Carl Bruck created a diagnostic test for syphilis using blood samples subjected to a complement-fixation test based on chemical processes newly understood by immunology.[86] Paul Erlich and Sahachiro Hata created the arsenical treatment Salvarsan (arsphenamine) in 1909.[87]



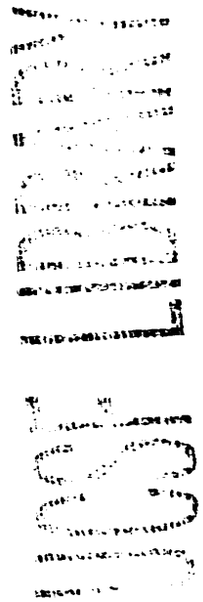
Did this new diagnostic technology and the new understanding of syphilis make the differential diagnosis of syphilis of the central nervous system and multiple sclerosis easier? The answer, in short, is not much. The Wasserman test had little effect during the 1910s and 1920s in simplifying the differential diagnosis. We can see why this might be so in the following examples. In 1912 F.X. Dercum, Professor of Nervous and Mental Diseases at the Jefferson Medical College in Philadelphia, related the case of a patient he had diagnosed as paretic during life but at the autopsy the "microscopic examination of the brain and of the cord disclosed the fact that the patient had not suffered from paresis but from multiple cerebrospinal sclerosis."[88] During the patient's life Dercum "did not at the

time have the opportunity of having a straight Wasserman made."[89] Dercum noted "that the mental symptoms of multiple cerebrospinal sclerosis may simulate paresis is of course well known."[90] Elaborating on the case Dercum remembered "the fact that in the present case the mental symptoms were pronounced from the outset, that they consisted of a rather pronounced mental loss, a dementia, together with a decidedly expansive mental state, suggested the diagnosis of a paresis. The tremor of the lips, tongue ataxic tremor, so frequently seen in multiple cerebrospinal sclerosis, the two attacks of hemiplegia which suggested the apoplectiform attacks of an early stage of paresis, the fugacious character of some of the symptoms, the inconstant character of the nystagmus, the inequality and irregularity of the pupils, the fact that the light reaction became impaired in one pupil relatively early while the reaction to accommodation persisted-all these facts tended to confirm or were in harmony with the idea that the patient was suffering from paresis."[91]

Another reason the new diagnostic technology did not make the differential diagnosis between syphilis and MS easier was because even with a negative Wasserman neurologists continued to diagnose syphilis of the central nervous system. Tom A. Williams urged why this should be so in 1914 arguing that "it cannot be too often insisted upon that the absence of the reactions which are detected by the usual laboratory tests for syphilis, is by no means conclusive of the absence of that disease; the failure to find them merely indicates that, at that particular moment the patient is not reacting strongly enough in that particular way." He opposed the fashionble over-reliance on the "clinical laboratory." He did so because "a non-reaction pupil, an absent knee-jerk, a positive great toe sign, are no less objective than blood cell count and diazo reaction, or an Abderhalden test; furthermore, these latter are much richer in liability of false interpretation, as well as errors in observation than are clinical signs in the hands of an experienced neurologist."[92]

In 1921, at a conference of the Association for Research in Nervous and Mental

Diseases, neurologists discussed the value of the new laboratory technologies in diagnosing MS. Harvard neurologists James B. Ayer and Harold E. Foster pointed out that: "a review of the literature up to 1909 fails not only to reveal findings pathognomonic of the disease, but shows that many authors regard the fluid as essentially normal;"[93] and that "no writer claims changes which can be construed as indicative of multiple sclerosis, certainly not pathognomonic."[94] Ayer's and Foster's own analysis of 38 M.S. patients showed a negative Wasserman in every case.[95] However, in revision of previous studies on the cerebrospinal fluid of MS patients Ayer and Foster argued that perhaps the new gold sol curve might have some "clinical significance."[96]



The problem was that the gold sol curve suggestive of MS could also indicate those very conditions likely to be confused with MS. Further, as Ayers and Foster noted, the progressive cases of MS of long-standing were most likely to have the paretic gold sol curve. These were the type of MS cases more easily diagnosed in the clinic; so the lab tests were of little value to the average clinician and did not make the differential diagnosis easier. At this same conference Bernard Sachs remained very skeptical of the value of the new laboratory technology. In a discussion following his paper, Sachs emphatically argued that "so far as the biological tests are concerned, perhaps I take a rather extreme view of the matter. I never allow biologic tests largely to influence diagnosis. If I make up my mind on clinical grounds that a case is multiple sclerosis, I am willing to have the laboratory corroborate that diagnosis, but I will not allow the negative findings to upset the positive diagnosis. I still maintain there may be lues in spite of negative findings."[97]

Physicians and neurologists usually suspected syphilis of the central nervous system first when a patient presented polyphonic clinical symptoms. Syphilis, as a disease category, acted as a filter through which doctors read the patients' bodies before them in the clinic. Syphilis was the default diagnosis. The presumption of syphilis was often so great

that physicians would prescribe Salvarsan despite negative laboratory tests. For example, examine this case: a 28 year-old Irish elevator guard appeared at the New York Hospital on March 22, 1919 complaining of a "tingling sensation in both legs and his right arm." For the past two months he had experienced difficulty walking, twitchings, paresthesia, and was very constipated. The physician noted on his admission form that the patient was a "married-ex-soldier-formerly a machinist. No work now-because of his illness. Has one child. Wife only pregnant once. Denies gonorrhea and luetic infections. Smokes one package of tobacco a week. Drinks glass of beer occasionally."[98] This physician gave a provisional diagnosis of "Cerebral-Spinal Syphilis."[99] On March 25, 1919 the patient's blood and spinal fluid analyses revealed a negative Wassermann test and a negative colloidal gold test. His urinalysis was also negative. In spite of the negative Wassermann physicians placed the patient on Salvarsan on April 3, 1919. On April 11, 1919 Dr. <name withheld> from the Department of Neurology at Columbia University, gave the patient a neurological exam and reported: "positive signs at present are: slight lateral nystagmus, in both directions, possibly more marked to right; astereognosis, right hand; diminished abdominal reflexes, equal; markedly increased knee jerks, right greater; slight spasticity of legs; positive Babinski, Oppenheim, and Gordon, both legs but more of right-not constantly obtained; moderately positive Romberg; slight incoordination, right leg and right arm; points by to the right with right hand. Possibly a beginning multiple sclerosis; some organic central nervous lesion at least."[100] Physicians only entertained a diagnosis of MS after an expert consultation from Columbia University. Most patients in the United States did not have access to expert university consultations in the 1910s and 1920s. Lack of access to expert neurologists meant that many patients, who would later be diagnosed with multiple sclerosis, received a diagnosis of syphilis of the central nervous system.

To get a better idea of how easy it would have been for syphilis of the central

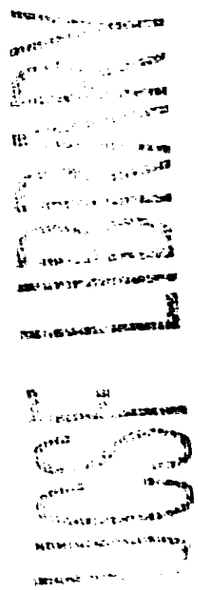
nervous system to be the first diagnosis which would have come to mind in a neurological clinic, as well as the general clinic, note the following statistics given by Bernard Sachs and Emanuel D. Friedman in 1921:

Comparative Incidence of Multiple Sclerosis and Lues of the Central Nervous System at Montefiore Hospital for Chronic Diseases, 1914-21 Inclusive:

MS	50
Lues of the central nervous system	167
(a) Cerebro-spinal lues	65
(b) Tabes	85
(c) General Paresis	17

At Mt. Sinai Hospital, 1919-21 Inclusive

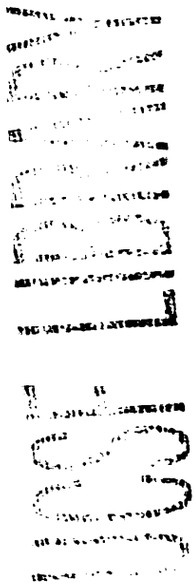
Total admissions to the neurologic service	2,357	
MS	90	(3.8%)
Lues of the central nervous system	562	(23.8%)
(a) Cerebro-spinal lues	317	
(b) Tabes	160	
(c) General Paresis	85[101]	



Syphilis of the central nervous system was the most common disease neurologists saw in the 1920s. This created a prejudice toward diagnosing the condition even though many of the cases might have been alternatively read as MS. Again, this was not just a problem for the less-experienced general practitioner, but also for the well-trained neurologist.[102] The fluid boundaries between syphilis and MS continued into the late 1920s. For example, in July of 1926, a 36 year-old Italian laborer presented himself at the New York Hospital displaying difficulty in walking, shaking hands, and weakness in his knees and back. Other doctors had previously diagnosed syphilis and alcoholism. A New York Hospital physician diagnosed disseminated sclerosis with syphilis of the CNS as a complication. He did this even though the "the blood and spinal fluid Wassermann's were negative for Lues on two occasions but the spinal fluid contained 79 cells per c.mm. with 80% lymphocytes on the first examination and 344 cells per c.mm. with 84% lymphocytes

on the second exam . . . In spite of these findings he was put on anti-luetic treatment."[103] The elevated cells in the spinal fluid could have indicated an infection of some sort to the physicians other than syphilis but they do not seem to have entertained this because multiple cerebro-spinal syphilis was the most common disease seen to affect the central nervous system and was the default diagnosis.

This can be seen again in this example from May of 1927: New York Hospital physicians saw a 44 year-old male mechanic from Brooklyn whose nationality the recorder listed as U.S. Protestant. The patient complained of an unsteady gait. The admission form recorded that the "Patient was in the Kings County Hospital the first 2 weeks of Sept. 1926 and in the Welfare Island Hospital from the 28th of Sept. 1926 till Dec. 23, 1926 for this sickness. There the differences of opinion seemed to be between Tabes Dorsalis and Multiple Sclerosis and he received K.I. <potassium iodide> for treatment blood and spinal fluid tests, presumably Wassermann, were negative according to the patient."[104] A neurological consultant gave the patient a diagnosis of disseminated sclerosis. Nevertheless, "the patient was given anti-luetic treatment on the chance that it might improve his condition. He went home given 30 grains of potassium iodide."[105]



Syphilis remained the default diagnosis for patients in the neurological clinic who presented protean clinical symptoms in the late 1920s and 1930s. Charles S. Potts and R.L. Drake remarking on this in 1928 wrote that "there is too great a tendency among practitioners of medicine to ascribe every organic disease of the nervous system to syphilis. This is far from the truth. Even a positive Wassermann is not proof that the condition is due to syphilis if the clinical symptoms are not present. This not infrequently leads to erroneous diagnosis and improper therapeutics."[106]

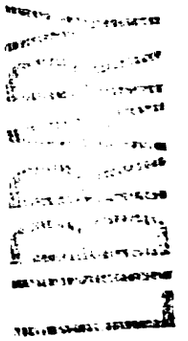
Physicians still frequently diagnosed with syphilis patients who later might have been diagnosed with MS in the 1930s as this case shows: "In 1937, P82 had what

appeared to be spasmodic torticollis. I referred her to Dr. #21 (deceased), in San Antonio, Texas, and after examination of her spinal fluid, he diagnosed cerebro-spinal lues; however, his diagnosis was based on a ONE-PLUS <caps in orig.> Wassermann reaction and a slight elevation in the first four components of the colloidal gold test. This spinal fluid was not checked by another laboratory, nor was a second specimen of spinal fluid obtained. . . So, doctor, that is all I have to offer. In my opinion, she did not have cerebro-spinal lues, and that tryparsamide therapy was unnecessary."[107]

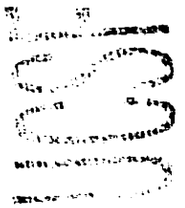
By the late 1940s syphilis of the central nervous system had declined as a default diagnostic category for neurologists. In 1948 Foster Kennedy remarked on how things had changed with regard to syphilis and MS; he said that "forty years ago anyone with 'nervous' legs was said to have locomotor ataxia, now he is quickly called 'multiple sclerosis.' Such diagrams save thought, on the part of those who have little thought to spare. The results may be life tragedy."[108] This change had not come through increasing reliance on laboratory technology in diagnosis because it remained of equivocal help in the 1940s and 1950s; rather, the change had come through the decline of secondary and tertiary syphilis generally. The decline of syphilis of the central nervous system as a default category, and the emergence of MS out of syphilis was a significant source of the perception of the rise of MS as a disease problem by neurologists. MS bubbled to the top as a disease category of importance as syphilis declined and neurologists were more likely to diagnose those patients suffering demyelination as multiply sclerotic rather than syphilitic. When a patient came to the clinic in the 1950s with a protean symptomatology, neurologists filtered these signs through the disease category of MS first rather than syphilis as they had done in the 1930s and before. This contributed substantially to the rise of MS cases in the United States.

The decline of hysteria as a disease category was another large nosological category from which MS cases migrated from the 1870s to the 1950s. Here is one early 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 October 20, 1873. Seguin reported that the patient was "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 . . . Ever since she has had weak right leg, without anaesthesia or numbness . . . almost cured once or twice; of late has required help of crutch, or friend's arm in walking . . . 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 acting upon her, I concluded that the patient had a functional palsy of an hysterical nature."[109] This patient died on August 1, 1874 and the autopsy revealed "disseminated sclerosis of the spinal cord."



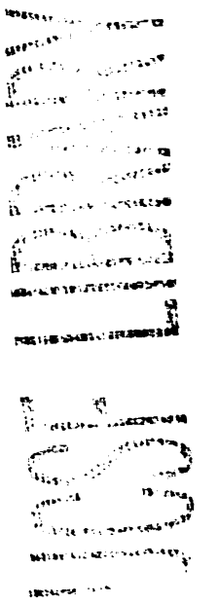
American physicians began increasingly to write about problems they were having with the differential diagnosis of hysteria from MS in the medical literature of the late 1890s. For example, Bernard Sachs wrote in 1898 that "we may concede that hysteria may simulate multiple sclerosis. More often hysterical symptoms are present in addition to those of multiple sclerosis. The differentiation will depend largely upon the presence of such distinctly hysterical stigmata as are foreign to the pure type of multiple sclerosis."[110] Also in 1898, Charles E. Beevor taught that "the diagnosis from hysteria is of the greatest importance, and it is often very difficult and sometimes impossible . . .." [111]



During this time neurologists began to write on how the discriminating clinician could differentiate between hysteria and MS. In 1899 Frank P. Norbury taught that in MS "the flexor muscles of the legs" assume "almost a spastic condition. This symptom is

pathognomonic of multiple sclerosis and cannot very well be imitated by hysteria."[112] He added that intention tremor, nystagmus, slow onset of the disease, cerebellar gait, spastic weakness of lower extremities, syllabic speech; exaggerated tendon reflexes and ankle clonus constituted "a group which pronounces the disease of organic origin and eliminates hysteria by the presence of ankle clonus, exaggerated reflex and absence of plantor <sic> reflex."[113]

In 1903 a family physician sent a thirty year-old laundryman to Drs. John Green, Jr. and Sidney Schwab because he found the diagnosis perplexing. Green and Schwab used the case to illustrate an aspect of the difference between hysteria and multiple sclerosis: "in the differential diagnosis between multiple sclerosis and hysteria the ocular symptoms will often give important aid. Changes in the optic papilla with transitory central scotoma, recession of the color fields in their physiological order, and the presence of nystagmus and paralyses, speak for multiple sclerosis. On the other hand, a normal disk, contracted fields without central scotoma, irregular and erratic contraction of the color fields, and the absence of oculo-motor disturbances would all tend to corroborate a diagnosis of hysteria."[114]



However, the following examples show how difficult the differential diagnosis between hysteria and MS remained for even elite neurologists in the early twentieth century despite increasing clinical acumen. In 1903 in the discussion following a paper presented at the Medical and Chirurgical Faculty of Maryland meeting at the Clinical Medicine Section held on February 6, 1903, Dr. Osler remarked on the relationship between hysteria and the apparent infrequency of MS: he said that: "Bramwell recently commented on the greater frequency of multiple sclerosis in England than in the United States . . . We may mistake the disease for hysteria unless we have read Buzzard's article. The ordinary typical cases are very rare."[115] On February 20, 1903, James Jackson Putnam discussed a

confounding case in a Clinical Meeting of the Staff of the Massachusetts General Hospital: "the other patient exhibited in the course of her illness a great variety of morbid signs and symptoms, quite sufficient, as I now think, to have established the diagnosis. Nevertheless, for some years before her death she was in a condition of well-marked spastic paralysis, which confined her to her bed and chair, and threw all other symptoms into the shade. This case is interesting from another point of view. It has, namely, been recognized that one of the diseases for which insular sclerosis may be mistaken is hysteria, and that diagnosis suggested itself several times to persons who examined this patient during the early part of her illness . . . " Her autopsy proved multiple sclerosis.[116]

In 1908 Daniel R. Brower wrote that "hysteria is the disease with which it <MS> is most frequently confounded" and Peter Bassoe "pointed out that cases of multiple sclerosis often for years are considered to be hysteria on account of the rapid and irregular appearance and disappearance and great variability of the symptoms."[117] In 1910, Theophil Klingmann emphasized the difficulty and importance of the relationship between MS and hysteria: "in the symptomatology this organic disease has two general conditions in common with the functional disorders of the nervous system and especially with hysteria, namely, the peculiar combinations and transitory character of symptoms which are in their anatomical distribution apparently unsymmetrical and unsystemic. Hysteria is probably the most important functional disorder to consider in the differential diagnosis."[118]

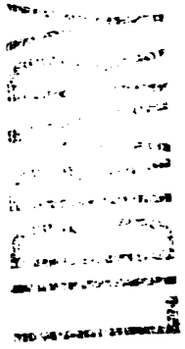
In 1914, Joseph Collins, physician to the New York Neurological Institute, and Edmund Baehr, Junior Neurologist to the Cincinnati General Hospital in Ohio, believed that they had "not encountered the difficulty said to exist, in distinguishing between disseminated sclerosis and hysteria." However, Collins and Baehr conceded that "the majority of authors, however, lay considerable stress upon the similarity between them. They are practically of one voice, furthermore, in saying that the difficulty generally results

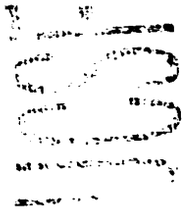
in mistaking an existing disseminated sclerosis for hysteria. The two disorders are common to youth and young adult life, and both frequently develop after a physical or psychical trauma. A widespread variability of disorders is common to both of them, involving somatic, visceral, and mental functions."[119] They then proceeded to teach the reader how to distinguish between the two diseases: "optic pallor and optic atrophy of hysterical nature is impossible. True nystagmus should always be considered evidence in favor of the organic disease . . . Sharply defined anesthesias and analgesias are not usually found in disseminated sclerosis and an anesthetic cornea practically never occurs. Bladder disorders are usually indicative of disseminated sclerosis, and actual exaggeration of the reflexes, especially when accompanied by a Babinski sign, must always be regarded as proof of organic nervous disease."[120]

These multiple signs, even ocular ones, could easily mislead the unwary. In a discussion which followed Foster Kennedy's paper on the subject at the Section on Nervous and Mental Diseases at the 65th Annual Session of American Medical Association in Atlantic City, New Jersey in June, 1914, Dr. Peter Bassoe of Chicago asked: "will Dr. Kennedy state the frequency with which optic neuritis occurs in the early stages of disseminated sclerosis? I am inclined to think that there is a stage of optic neuritis in many of these cases before the familiar pallor of the disks sets in. I recall a case of a young woman with no physical signs of organic disease who suddenly became blind in one eye. There was a distinct hyperemia of the disk, but an able ophthalmologist stated that it was not necessarily beyond physiologic limits. The patient was treated for hysteric blindness and recovered her sight, but a year or so later she developed the ordinary signs of disseminated sclerosis."[121]

Three years later in 1917 the differential diagnosis between hysteria and MS continued to be a problem as this discussion following the paper of Leo M. Crafts of

Minneapolis, at the Section on Nervous and Mental Diseases, at the 68th annual AMA meeting held in Cincinnati shows: Dr. D. I. Wofstein of Cincinnati said that "often there is difficulty in distinguishing between this disease and hysteria, but if one keeps a sharp lookout for organic signs, such as a Babinski sign or mild clonus, or slight rigidity, the proper diagnosis may be made."[122] Dr. G.A. Moleen of Denver then pointed out that it was not quite as easy as that: "there is one point which was omitted in the paper and that is the frequent mistaking of this condition for hysteria . . . In this condition we make many mistakes . . . He was often forced to revise his diagnosis of hysteria. The characteristic striking remissions after very grave symptoms are misleading. This would suggest hysteria, but is, after all, a characteristic of this disease"[123] Dr. E.D. Fisher of New York concurred saying that "the differential diagnosis between multiple sclerosis and hysteria is often difficult to make. Many apparently hysterical cases turn out to be multiple sclerosis."[124]

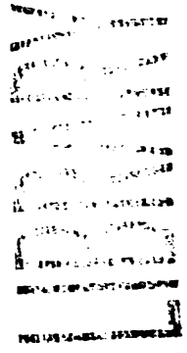


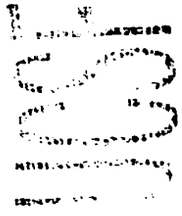


The differential diagnosis of MS and hysteria remained a difficult clinical problem at the Association for Research in Nervous and Mental Diseases meeting held on MS in 1921.[125] Boston physician Maurice Fremont-Smith presented a case in 1927 to demonstrate the difficulties one encountered in private practice with regard to this question.[126] He outlined the case history of a "well-developed and well-nourished woman of thirty-five, nervous and mentally depressed." The patient's "pupils were equal and reacted to light; ocular motions were normal; the fundi were negative. There was no cranial nerve paralysis . . . The keen-jerks were equal and moderately active, ankle-jerks equal and normal. Biceps and triceps normal. There was no clonus and no Babinski. Vibratory and toe position sense were normal. There was not ataxia of legs. Romberg was negative. Sensation for pain and touch was normal. Blood Wasserman was negative." Fremont-Smith wondered if he was "dealing with a nervous, poorly adjusted individual, with

hysterical manifestations, or with a degenerative condition of the central nervous system?" He diagnosed her with hysteria "because of the glove-like distribution of her sensory symptoms ('numbness in both feet, less marked in legs up to knees, back and front'), the lack of confirmatory evidence on objective examination, the entire absence of objective evidence pointing to organic cord or blood disease, and last, her emotional reactions during examination, which were manifestly of the hysterical type."[127]

In January, 1926, nine months later, Dr. George Clymer found that the speech of this same patient was scanning, "she had a slight intention tremor, nystagmus, double Babinski, and suggestive though unsustained ankle clonus. I think there is no doubt at present that the diagnosis is multiple sclerosis."[128]

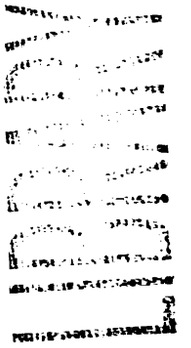


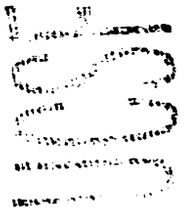


In the 1930s hysteria and MS still shared a blurry nosological boundary in clinical practice. Paul De Nicola presented a case before the New Hampshire Medical Society at Manchester on May 16, 1933. In the discussion following the paper, Dr. Charles H. Dolloff of Concord, NH said that "one of the interesting phases of this disease, so far as the psychiatrist is concerned, is the possibility of its being mistaken in its earlier stages for hysteria."[129] This is very important if one remembers that most physicians during the 1930s concerned with neurology were diplomates in neurology *and* psychiatry. Further psychiatry at the time was more prestigious and had more professional power than neurology. This led many psychiatric/neurological practitioners to see their patients more through a psychiatric than a neurological lens. The diagnostic category first in the minds of the psychiatrically inclined when a patient presented protean and transitory symptoms was often hysteria. They expected to see hysteria and so they often did. Lewellys F. Barker in a Clinic for Senior Students of the Johns Hopkins Medical School emphasized this in 1936: "a mistake far too often made by practitioners on seeing a patient with multiple sclerosis in its early stages is to assume that the symptoms are due to hysteria; the phenomena observed

are thought to be of functional origin, especially when the disease undergoes a marked remission. In many instances in which a diagnosis of hysterical amaurosis or of functional hysterical dysbasia has been made, a more careful study would have shown the organic nature of the process."[130]

The differential diagnosis between hysteria and MS still posed a problem in the 1940s. During World War II it was a problem for air surgeons as can be seen from this case reported by George J. Wayne of the Carlsbad Army Air Field in New Mexico: "this case is of interest because it presents the well-known problem of differential diagnosis between conversion hysteria and an organic neurologic lesion <MS>. Because the symptomatology suggests the possibility of conversion hysteria, representing a rejection of flying and its associated dangers, it is of special interest to the flight surgeon. The case emphasized the importance of performing a thorough psychiatric inventory and neurologic examination before final diagnosis."[131]



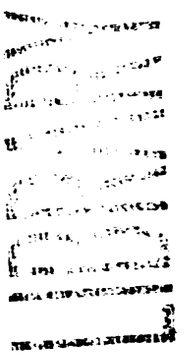


The differential diagnosis between hysteria and MS still posed a problem in the 1940s.[132] However, the newly formed National Multiple Sclerosis Society indicated that in the late 1940s the diagnosis of multiple sclerotics as hysterical was declining.[133] Nevertheless, a New York Neurological Institute neurologist still maintained in 1948 that it was still of concern especially in the early stages of the disease.[134] In the 1950s physicians continued to diagnose many MS cases as hysterical; however, the frequency of this seems to have declined somewhat.[135] In short, the gradual decline of hysteria as a diagnostic category led to increasing numbers of MS diagnoses in the United States.

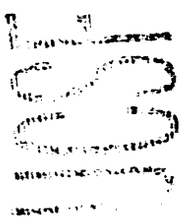
Like syphilis, hysteria declined as a diagnostic category during the first six decades of the twentieth century. Mark S. Micale has argued that hysteria did not disappear so much as migrate into a multitude of new nosological categories after the late nineteenth century. The most important of these categories included: syphilis, epilepsy, various

German psychotic categories and Freudian psychoneuroses. Micale only mentions multiple sclerosis in passing.[136]

We can understand better how important the decline of hysteria as a diagnostic category was, despite its persistence, to the rise of MS as a diagnostic category if we understand the function of gender in the clinical encounter of diagnosis. Physicians were much more likely to diagnosis women with hysteria than men in Europe and North America. Throughout the nineteenth century in Europe and North America, doctors considered hysteria the most common neurological/psychiatric condition that affected women between menarche and menopause.[137] For example, New York Hospital physicians diagnosed 126 men with hysteria and 620 women with the same condition from 1878 to 1906 excluding 1894.[138]



As hysteria gradually declined as a neurological diagnosis in the first six decades of the twentieth century, physicians interpreted increasing numbers of these patients, especially women, as multiple sclerotics. We can see this by studying the changing perception of the sex differences in MS statistics. From the 1870s to the 1910s some American neurologists considered MS to affect women slightly more than men; others considered men to be slightly more afflicted; while many held that the sexes were equally affected. None had sufficient statistics to make more than a guess based on their own experiences.[139]



To remedy this, for the 1921 Association for Research in Nervous and Mental Diseases (ARNMD) meeting devoted to MS in New York City, Israel S. Wechsler analyzed the largest sample ever studied for this purpose, 1,970 patient records. Wechsler concluded that "the male is more often affected than the female, in the ratio of nearly 3 to 2."[140] On reviewing Wechsler's data the Commission of the ARNMD concluded Wechsler was correct.[141] Thus, the orthodox view in 1921 was that MS affected the men more than

women by a ratio of 3 to 2.

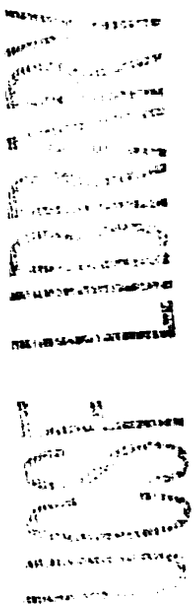
During the rest of 1920s through the 1940s the journal literature showed differences of opinion regarding the sexual statistics among MS patients. Some held that the disease affected the sexes equally while others maintained that MS affected women slightly more than men. Again, most physicians based these conclusions on anecdotal reports from their own clinics. However, during this period, there was a general perceptual shift toward believing that MS affected men and women equally. Few if any neurologists thought men more affected by the 1940s.[142] In corroboration of this, in the largest sample since the 1921 meeting, Charles C. Limburg, at the 1948 conference of the ARNMD, also devoted to MS, concluded that MS afflicted men and women equally.[143] During the 1950s the official word from the National Multiple Sclerosis Society was that the disease affected the sexes equally.[144] More exhaustive epidemiologic studies during the early 1950s, conducted under the auspices of the Public Health Service, showed that MS occurred perhaps to "a greater extent in females."[145] Reviewing the question in 1960 George Schumacher noted that: "most analyses suggest a slightly higher incidence in women."[146] To recapitulate, in 1921 the orthodox view was that MS affected men more than women by a ratio of 3 to 2. In 1948 the official view was that MS afflicted men and women equally. By 1960 neurologists generally thought MS affected women more than men. This trend continued so that by 1993 the view was that MS beset women twice as often as men.[147] So, physicians increasingly diagnosed women, at least after 1921, as multiple sclerotics. What accounts for this gendered epidemiological shift over time?

Because of the gendered filter through which neurologists read and interpreted the symptoms generated during the clinical encounter between the physician and patient, doctors were more likely to diagnose men with multiple sclerosis than women even if they had identical symptoms even in the 1950s. There was a greater prejudice to see transitory

and polyphonic symptoms in men as organic while these same symptoms in women were more often interpreted as hysteric.

As the diagnosis of hysteria declined and dispersed after its apogee in the late nineteenth century, physicians reinterpreted as multiple sclerotics increasing numbers of patients, especially women, throughout the twentieth century. This accounts for a share of the rise in the perception of increasing numbers of MS patients especially between the 1920s and the 1950s and after.

VI Conclusion



The changing prognosis in terms of longevity after diagnosis from the 1870s to the 1950s helps us see the extent to which American physicians diagnosed MS earlier and earlier in the interpretive process. In the late nineteenth century neurologists estimated life expectancy for multiple sclerotics at two to ten years from diagnosis.[148] By 1954, based on more experience and better data, neurologists saw the average life expectancy after onset to be approximately twenty-one years which represented a two to three fold increase from the late nineteenth century.[149]

However, American neurologists did not just see MS earlier but saw it as MS instead of something else such as syphilis or hysteria. Physicians recognized MS earlier because the ongoing specialty formation of neurology meant that more physicians had the skills necessary to see MS through time and because alternative diagnostic categories, such as hysteria and syphilis of the central nervous system, declined throughout the twentieth century. This meant that neurologists were quicker to interpret the polyphonic symptomatology of demyelinated patients as MS. These reasons do not rule out that there were more cases of MS due to an actual biological increase in the underlying pathophysiology of demyelination in the population. They do, however, significantly complicate any attempt to demonstrate this.

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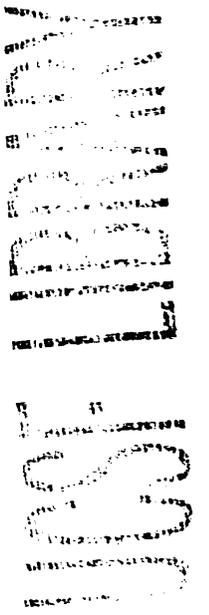

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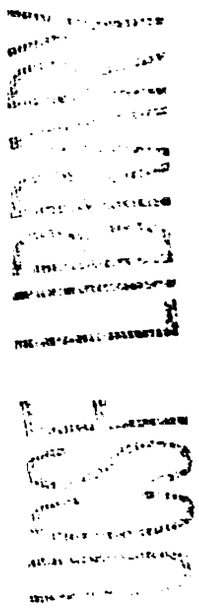

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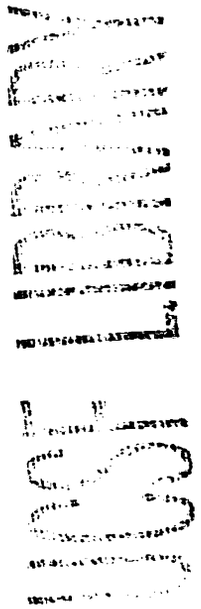

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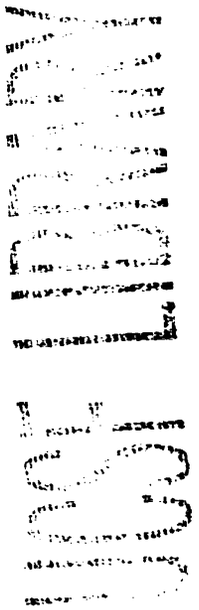
> certain undeveloped forms occur, simulating various nervous diseases. They are known as the *formes frustes.* On account of the gait, ataxia, tremor, nystagmus, and speech defect this disease many be confounded with the several hereditary ataxias. Friedreich's ataxia can be excluded by the presence of knee-jerk with clonus and optic atrophy and absence of sclerosis and contractures. The Romberg phenomenon, transitory oculo-motor palsy, absence of family history, and peculiar optic atrophy, differentiate the picture from a hereditary cerebellar ataxia. Syphilis of the brain and cord can be ruled out by the absence of history and signs of the disease, and the failure of the therapeutic test. Brain tumor of the posterior fossa is sometimes accompanied by this form of gait, ataxia, and nystagmus, but the more rapid progress of this condition, the greater degree of headache and vertigo, and the positive symptoms of multiple sclerosis, present no difficulty in eliminating tumor. In the adult the tumor may cause confusion with Parkinson's disease, but from the totally different character



of the tremor, the age, attitude, mask-like face, and rigidity, with the tendency to festination, no errors should be made. Certain cases of paralytic dementia present symptoms of tremor, peculiar gait, and speech disturbance, but the alteration in character and disposition, the fine fibrillary tremor of the tongue and . . . the Argyle-Robertson pupil, the stumbling over and dropping of syllables, and the more marked mental deterioration, should prevent the confusion of these diseases.

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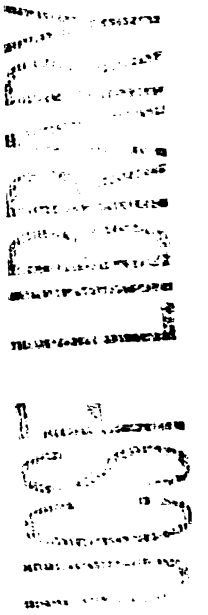
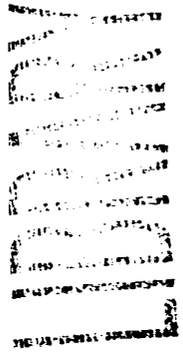
Mr. D. was examined by us at the Home for Incurables, in September, 1899. We saw him once only. He stated that he was forty-six years old, single, and with no vicious habits. He denied having used alcohol even moderately. His health previous to his present illness had always been good and he had never had any venereal disease. The onset of his present trouble was slow; its course progressive and steadily downward. Seven years ago he began to have difficulty in walking, and now cannot move his legs at all. He has not been able to walk or even stand for several months. He has never had any pain in the legs. He has, at times, had slight spasmodic retention of urine, but has never been catheterized. During the past year his bowels have been moved only after the use of purgatives. The necropsy revealed <MS> . . . He died suddenly a few weeks after our examination . . . We have, in short, a case which, during a course of seven years, presents the clinical picture of a posterior sclerosis; of an ataxic paraplegia (postero-lateral sclerosis); and immediately before death of spastic paraplegia; the typical symptoms of multiple sclerosis never at any time being present . . .That a disease process so extensive and irregular in its involvement of nervous tissue, so irregular in its etiology and development, should fail to follow any one clinical type is rather to be expected than otherwise . . . The diagnosis in our case was made difficult not only by the entire absence of what are usually considered the typical symptoms of multiple sclerosis . . . but also by the course of the disease itself, which, by picking out certain system tracts of the cord at successive intervals, led to the diagnosis by different observers of the disease suggested by the tracts affected. The diagnosis of tabes dorsalis made at the onset is easily explained by the pathological examination. A dense and by far the oldest plaques of sclerosis is found in the dorsal region and is almost entirely limited to the posterior columns. . . . A differential diagnosis in this stage would certainly be very difficult . . . With involvement of the lateral tracts spasticity was added to the other symptoms and under a different observer ataxic paraplegia was diagnosed. The lesions in the cervical cord show a much more advanced degree of sclerosis than the dorsal or cerebral lesions, and it was probably these lesions which caused the symptoms . . . . When the case came under our observation the loss of power was so extensive and the spasticity so great that it was impossible to determine any ataxia even had it been present. Unaware of the previous diagnoses, and in the absence of sensory or other localizing symptoms, a provisional symptomatic diagnosis of spastic paraplegia was made.



51. ARNMD, Discussion of Sachs and Friedman, "General Symptomatology . . ." *Multiple Sclerosis* (1922), 57-58. Sachs full response is as follows:

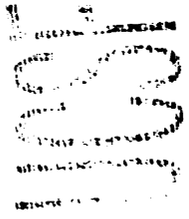
> There is no one symptom, nor are there any two symptoms that I would consider pathognomonic of the disease. You cannot base a diagnosis of this disease on any one or two symptoms. No doubt many of the old cases of primary or lateral sclerosis come now under the heading of disseminated sclerosis. If 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 at this point at least the first 9 groups of symptoms and try to make the diagnosis, if any number of these are present.



52. David A. Freedman and H. Houston Merritt, "The Cerebrospinal Fluid in Multiple Sclerosis," in Multiple Sclerosis and the Demyelinating Diseases, *Proceedings of the Association for Research in Nervous and Mental Diseases, December 10 and 11, 1948, New York City* (Baltimore: Williams & Wilkins Company, 1950), 428-439.

53. Ibid., 429 .

54. Ibid., 436. Putnam's full response was as follows:



> I am very glad to hear the authors call attention to the difference in incidence of colloidal gold curves obtained by minor difference in technic. This was a matter that troubled me very much on moving from Boston to New York. In our laboratory at the City Hospital we used to have a very high incidence of abnormal colloidal curves in cases of multiple sclerosis. If I remember rightly it was something like 60 or 70 per cent. At the Neurological Institute, on the other hand, the incidence was very much lower. My guess is that it was around 10 or 20 per cent. Inquiring into the discrepancy, I found the laboratory at the Presbyterian where the colloidal gold curves were performed were using the Army technic, which is so adjusted as to give as few positive reactions as possible outside of cases of neurosyphilis. It is studiously constructed so as not to give indications of multiple sclerosis, or other indications of multiple sclerotic conditions, or a minimum proportion. This is, I believe, the customary way of doing the test in most laboratories in this country.

55. Ibid., 436.

56. Ibid., 438. Merritt's full statement was as follows:

> I think it has been emphasized by Dr. Freedman and also by Dr. Von Storch that there is no specific finding in the cerebrospinal fluid that will make the diagnosis of multiple sclerosis. The colloidal gold, when it is properly performed, will be positive in a very high percentage of the cases, but the presence of the characteristic first-zone gold sol curve or Dr. Lange's D.

curve is not diagnostic of multiple sclerosis, although it is of helpful benefit in a case in which the diagnosis is clinically suspected. The difficulties in performance of the colloidal gold has been stressed by Dr. Putnam. His experience has been the same as mine. In going from laboratory to laboratory, you find a great deal of discrepancy. In Boston we were fortunate to work in laboratories that had been built up by Dr. Ayer and the technic for the colloidal gold was of a very high order. Dr. Lange and Dr. Harris, at Albany, have a technic that is very accurate. But if you will take the routine clinical laboratories throughout the country, I might not send them the fluid for colloidal gold, because, the percentage of positive are so low that the cost of the test is not justified. So that raises the question of whether another test would not be of more value

57. George A. Schumacher, "The Diagnosis of Multiple Sclerosis," in *The Status of Multiple Sclerosis,* ed. Roy Waldo Miner, *Annals of the New York Academy of Sciences* 58 (1954): 670.

58. Ibid., 573-74 .

59. Ibid., 574.

60. *Tracy Jackson Putnam Collection*, Louise Darling Biomedical Library, University of California, Los Angeles (Hereafter *TJP Collection*). University of California, Los Angeles Hospital Records (Hereafter UCLA Records).

61. Israel S. Wechsler, "Statistics of Multiple Sclerosis," in *Association for Research in Nervous and Mental Disease, Multiple Sclerosis [Disseminated Sclerosis], volume II* (New York: Paul B. Hoeber, 1922), 27. Albert G. Odell, "The Signs and Symptoms of Multiple Sclerosis with Particular Reference to Early Manifestations," *New York State Medical Journal* 31 (1931): 1018.

62. Meredith Clymer, "Notes on the Physiology and Pathology of the Nervous System, with reference to clinical medicine, Sclerosis of the Nervous Centres," *New York Medical Journal* 11 (1870): 232.

63. Bauduy taught that from "a diagnostic point of view it is important to bear in mind the following distinctions: Paralysis agitans is a functional disease of the nervous system dependent on a morbid condition of motor roots, and in which there is no paralysis. In multiple cerebral sclerosis tremor always precedes paralysis, is accompanied by festination, and the tremor is produced by movements whether voluntary or involuntary. In multiple cerebro-spinal sclerosis paralysis precedes tremor, and the latter is only developed during voluntary muscular movements; it is likewise accompanied by festination. In sclerosis of the antero-lateral columns there is no tremor, no festination," J.K. Bauduy, "Clinical Lecture Multiple Cerebro-Spinal Sclerosis," 5.

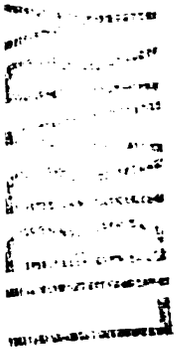
64. Allan McLane Hamilton, "Cerebro-Spinal Sclerosis," in his *Nervous Diseases: Their Description and Treatment* (Philadelphia: Henry C. Lea, 1878), 346.

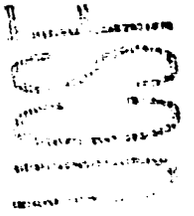
65. Hugo Engel, "Multiple Cerebro-Spinal Sclerosis and Paralysis Agitans," *The Medical and Surgical Reporter* 40 (1879): 357-60.

66. A. B. Arnold, "Multiple sclerosis of the Brain and Spinal Cord," *The Southern Clinic* 1 (1879): 258.

67. Theodore Diller, "An Atypical Case of Insular Sclerosis," *NY Med J* (5/25/1895): 644. F. X. Dercum, "Multiple Sclerosis: Traumatic tremor, Railway Spine," *International Clinics* 1 (1893): 122-128. This article was a clinical lecture delivered at the Philadelphia Hospital. F.X. Dercum, "Multiple Sclerosis; Traumatic Tremor, Railway Spine," (clinical lecture delivered at the Philadelphia Hospital), *International Clinics: A Quarterly of Clinical Lectures* 1 (1893): 122-123.

68. Onuf (Onufrowicz), "The Differential Diagnosis of Multiple Sclerosis,"





Brooklyn Medical Journal 16 (1902): 483-87.

69. Clymer, "Notes on the Physiology . . . ," 234.

70. 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.

71. William Krauss, "A Case of Disseminated Insular Sclerosis," *Memphis Medical Monthly* 19 (1899): 451-452.

72. Joseph Collins and Edmund Baehr, "Disseminated Sclerosis," *American Journal of Medical Sciences* 148 (1914): 515. Tracy J. Putnam, The Diagnosis of Multiple Sclerosis and the Outlook for Treatment," *Medical Clinics of North America* 21 (1937): 584. I Abrahamson, "Multiple Sclerosis ?" *JNMD* 29 (May 1902): 287-290.

73. I Abrahamson, "Multiple Sclerosis ?," 288. See also B. Onuf (Onufrowicz), "The Differential Diagnosis of Multiple Sclerosis," 484. Joseph Collins and Edmund Baehr, "Disseminated Sclerosis," 515.

74. From the discussion which followed Crafts paper presented to the Section on Nervous and Mental Diseases, American Medical Association, 68th meeting, June 1917, Dr. Bernard Sachs, 1136, in Leo M. Crafts, "The Early Recognition of Multiple Sclerosis," *JAMA* 69 (1917): 1130-37.

75. E.W. Taylor, "Multiple Sclerosis: The Location of Lesions with Respect to Symptoms," *ANP* 7 (1922): 580

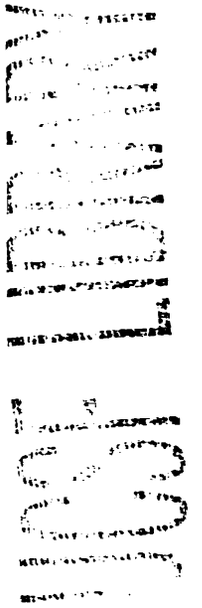
76. Tracy J. Putnam, "The Diagnosis of Multiple Sclerosis and the Outlook for Treatment," *Medical Clinics of North America* 21 (1937): 584. In 1937 George B. Hassin, Professor of Neurology at the University of Illinois College of Medicine and Attending Neurologist at Cook County Hospital, wrote that

> a combined lesion of the spinal cord and the optic nerve fibers is not uncommon. It occurs in multiple sclerosis, cerebrospinal syphilis, disseminated tumors of the central nervous system, encephalomyelitis, septicopyemia and so-called neuroptic myelitis (Devic's disease). Of the foregoing conditions, neuroptic myelitis has attracted a great deal of attention of late, and, as it usually runs an acute or subacute course (from two months to one year), it is often designated as acute multiple sclerosis or disseminated encephalomyelitis. For this reason it is generally not considered a specific disease process, though some view it as a well defined clinical entity, different from acute disseminated encephalomyelitis or so-called acute multiple sclerosis. The results of histopathologic study of a case seem to favor the latter view, that neuroptic myelitis is a well defined clinical syndrome with definite clinical features."

George B. Hassin, "Neuroptic myelitis versus multiple sclerosis," *ANP* 37 (1937): 1083.

77. #105, M.D. Asst. Resident in Medicine, Discharge Summary, U242, 6/5/58, UCLA Records. The full quote is as follows:

> Gait is shuffling with weakness in dorsiflexion of right foot and weakness of flexion of right hip. Patient is unable to walk on the toes of her right foot. She is not able to step onto a chair with right leg. In supine position the patient is unable to overcome gravity in flexing the right hip when elevating the right foot for dorsiflexion. Pos. Babinski on right and reflexes on the left are somewhat hyperactive. Light touch sense absent and stocking distribution below the left knee and in small patches in the right leg below knee. Temperature reception is absent below the left knee. Pin prick over the entire left leg creates a burning sensation which is not perceived in the



right leg . . . Following hospitalization the patient developed mild symptoms of urinary retention which had not been present prior to discharge. She was placed on Brewer's yeast tabs. 5 q.i.d. the patient was seen in consultation by Dr. #107 who felt that the patient had a myelitis of undetermined cause involving primarily the lateral fiber bundles on the right side at about the T-12 L-1 level. The possibility of early multiple sclerosis must be considered but can not be established without more definitive evidence of multiple lesions in the central nervous system.

See also, National Multiple Sclerosis Society, "Multiple Sclerosis Diagnosis and Treatment," *JAMA* 135 (1947): 569. Tracy J. Putnam, "Sclerosis and encephalomyelitis," *Bulletin New York Academy of Medicine* 19 (1943): 302. Richard M. Brickner, "Multiple Sclerosis," *Medical Clinics of North America* 32 (1948): 744-50.

78. Sanger Brown, "Diagnosis of Insular Sclerosis," *Illinois Medical Journal* 14 (1908): 201. During this time medical workers produced knowledge about syphilis and multiple sclerosis simultaneously. Alfred Fournier demonstrated the syphilitic origin of tabes dorsalis in 1875. See Claude Quétel, *History of Syphilis*, trans. Judith Braddock and Brian Pike (Baltimore: The Johns Hopkins University Press, 1992, original 1986), 134.

79. C.H. Boardman, "Progressive Multiple Cerebro-spinal Sclerosis," *The Northwestern Medical and Surgical Journal* 3 (1873): 258. See also, George S. Gerhard, "Cases of Multilocular Cerebro-Spinal Sclerosis," *Philadelphia Medical Times* 7 (November 11, 1876): 51. A. B. Arnold, "Multiple sclerosis of the Brain and Spinal cord," *The Southern Clinic* 1 (1879): 257-59.

80. Gerhard, 51. H.C. Wood, "Cerebral, Spinal and Cerebro-Spinal Sclerosis, a Clinical Lecture," *Michigan Medical News* 3 (1880): 171-72. See also, Charles K. Mills, "On Posterior Spinal Sclerosis," *Medical Gazette* 7 (1880): 1-3. F. Woodbury, "Diffuse Sclerosis of the Spinal cord and Medulla Oblongata-Disease of Freidreich," *Philadelphia Medical Times* 8 (2/24/1883): 372-375. Maurice L. Goodkind, "Multiple Sclerosis, Double Abducens Paralysis, and Locomotor Ataxia," *Medicine* 4 (1898): 184-189.

81. Sachs then instructed his listeners how they might make the differential diagnosis: "in casting about for further symptoms which should help us to distinguish between the two diseases in doubtful cases, I believe that the greatest stress should be laid upon ocular conditions. First of all, in syphilis of the brain, nystagmus is rare . . . ocular palsies occur in both affections, but they are rarely as complete in multiple sclerosis as in syphilitic affections . . . complete immobility of the pupils on exposure to light and during accommodation is more common in syphilitic affections than in any others . . . while primary optic atrophy, and particularly partial atrophy of the optic nerve, is much more characteristic of multiple sclerosis." Bernard Sachs, "The Relation of Multiple Sclerosis to Multiple Cerebro-Spinal Syphilis and to Paralysis Agitans," *The Philadelphia Medical Journal* 1 (1898): 241-44.

82. Bernard Sachs, "On Multiple Sclerosis, with especial reference to its clinical symptoms, its etiology and pathology," *JNMD* 25 (1898): 320, 326.

83. B. Onuf (Onufrowicz), "The Differential Diagnosis of Multiple Sclerosis," 484-85. The full quote is as follows:

> . . . cerebro-spinal syphilis . . . has many features in common with multiple sclerosis: First, the multiplicity of lesions. Second, the appearance in attacks separated by longer or shorter interals <sic> of relative freedom from new symptoms and of apparent stand-still of the disease. Third, the tendency of the symptoms to subside to a considerable extent . . . When all the cardinal symptoms of multiple sclerosis, the intentional tremor, the

> nystagmus, the scanning speech, the ataxy and the spasticity, are present, then probably the diagnosis will present no difficulties, but how often do we find only one or two of these present and frequently in a slight degree only, or atypically, as for instance the speech disturbance . . . The spasticity is just as frequently observed in syphilis, the ataxy, at least of the lower extremities, is also quite frequent in syphilis, and the intentional tremor, on which so much diagnostic reliance is placed as in favor of multiple sclerosis, I have seen very typically developed in a case of undoubted cerebral syphilis. The nystagmus, when fairly developed, speaks pretty strongly for disseminated sclerosis as against syphilis, but is by no means pathognomonic, and if any ocular defects are present, such as leucoma, corneal or strong refraction errors, it loses considerably in value . . .

84. J. Ramsay Hunt, "Multiple Sclerosis with Dementia: a contribution to the combination form of multiple sclerosis and dementia paralytica," *American Journal Medical Sciences* 126 (1903): 982.

85. Quétel, 140.

86. Allan M. Brandt, *No Magic Bullet: a Social History of Venereal Disease in the United States Since 1880* (New York: Oxford University Press, 1985), 40.

87. Quétel, 142. Sachs and Friedman, "The Differential Diagnosis," 133-136.

88. F.X. Dercum, "A case of multiple cerebrospinal sclerosis, presenting unusual symptoms suggesting paresis," *JAMA* 59 (1912): 1612.

89. Ibid., 1612.

90. Ibid., 1613.

91. Ibid., 1613.

92. Tom A. Williams, "Syphilitic Multiple sclerosis diagnosed clinically in spite of negative laboratory tests," *Boston Medical and Surgical Journal* 171 (1914): 527. His full comments were as follows:

> It cannot be too often insisted upon that the absence of the reactions which are detected by the usual laboratory tests for syphilis, is by no means conclusive of the absence of that disease; the failure to find them merely indicates that, at that particular moment the patient is not reacting strongly enough in that particular way. The results of previous reactions, as manifested by present clinical signs, furnishes a basis for a diagnosis in every way as potent and no more lacking in that objectivity which it is the fashion to claim preeminently for certain methods conducted in the clinical laboratory. A moment's reflection shows the fallaciousness of this claim; indeed, a non-reaction pupil, an absent knee-jerk, a positive great toe sign, are no less objective than blood cell count and diazo reaction, or an Abderhalden test; furthermore, these latter are much richer in liability of false interpretation, as well as errors in observation than are clinical signs in the hands of an experienced neurologist.

93. James B. Ayer and Harold E. Foster, "Studies on the Cerebrospinal Fluid and Blood in Multiple Sclerosis," in ARNMD, *Multiple Sclerosis,* 1922, 113.

94. Ibid., 114.

95. Ibid., 115.

96. Ibid., 117-18.

97. Sachs and Friedman, "General Symptomatology of Multiple Sclerosis," 61.

98. "Admission Form," 1, Patient 3, NYH/Cornell Archives.

99. Ibid., 2.

100. "Neurological Exam," 4/11/19, Patient 3, *NYH/Cornell Hospital Archives.*

101. Sachs and Friedman, "General Symptomatology of Multiple Sclerosis," 55.

102. In 1921, Bernard Sachs and Emanuel D. Friedman, at the annual Association for Research in Nervous and Mental Diseases meeting continued to teach that "there is one differential diagnosis of paramount importance, and to that it will be well to give full consideration. In a given case, is the patient suffering from multiple sclerosis, or from multiple cerebrospinal syphilis? In both diseases we are confronted with patients who have in the course of a few months or possibly a few years developed from very slight beginnings a more or less marked spastic weakness of the lower extremities associated with an increase in all the deep reflexes. In both diseases, there are very marked periods of remission and of exacerbation. The remissions are just as marked in the one as in the other disease," Sachs and Friedman, "The Differential Diagnosis, Course and Treatment of Multiple Sclerosis," in ARNMD, *Multiple Sclerosis*, 1922, 133, 136.

103. "Discharge Note," Patient 5, *NYH/Cornell Hospital Archives.*

104. Admission Form, 5/23/1927, Patient 6, *NYH/Cornell Hospital Archives.*

105. House Physician, "Discharge Note," 6/20/27, Ibid. For the boundary between syphilis and multiple sclerosis see also, Patient 2, September 1927, *NYH/Cornell Hospital Archives.*

106. Charles S. Potts and R.L. Drake, "The Diagnosis of Multiple Sclerosis with Special Reference to Changes in the Cerebrospinal Fluid and Abdominal Reflex," *Medical Journal and Record* 128 (1928): 73.

107. Letter, #22, M.D., Victoria, TX to TJP 6/26/49, Folder P82, Box 10, *TJP Collection.*

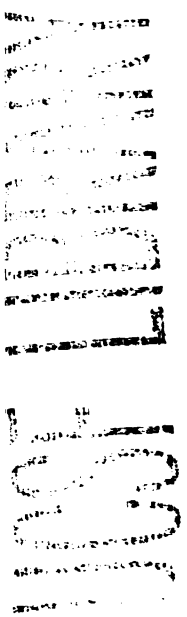
108. Foster Kennedy, "On the Diagnosis of Multiple Sclerosis," in ARNMD,*Multiple Sclerosis and the Demyelinating Diseases,* 1950, 526.

109. E.C. Seguin, "A Contribution to the Pathological Anatomy of Disseminated Cerebro-Spinal Sclerosis," 285-6. The full case is as follows:

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

110. Bernard Sachs, "On Multiple Sclerosis," 327.

111. Charles E. Beevor, *Diseases of the Nervous System a Handbook for Students and Practitioners* (Philadelphia: P.L. Balkiston, Son & Company, 1898), 276. See also,



Charles J. Aldrich, "Two cases of disseminated sclerosis and a case of jacksonian epilepsy of embolic origin, with presentation of cases." *Cleveland Journal of Medicine* 1 (1896): 455-58.

112. Frank P. Norbury, "A Case of Multiple Sclerosis and One of Cerebral Palsy in a Child," *Medical Herald* 18 (1899): 521.

113. Ibid., 522.

114. John Green, Jr. and Sidney I. Schwab, "Ocular Examination as an aid to the early diagnosis of multiple sclerosis, with report of a case," *Interstate Medical Journal* 10 (1903): 542.

115. Preston and Hirschberg "Case of Multiple Sclerosis," *Maryland Medical Journal* 46 (1903): 285.

116. J.J. Putnam, "Insular sclerosis; Charcot Joints," *Boston Medical and Surgical Journal* 149 (1903): 71.

117. Daniel R. Brower, "Diagnosis of Multiple Sclerosis," *The Archives of Diagnosis* 1 (1908): 265. Peter Bassoe, "Report of Four Cases of Multiple Sclerosis Complicated by Many Hysterical Phenomena," *Illinois Medical Journal* 14 (1908): 674.

118. Theophil Klingmann, "Visual disturbances in Multiple Sclerosis," *JNMD* (1910): 734.

119. Collins and Baehr, "Disseminated Sclerosis," 517.

120. Ibid., 517.

121. Foster Kennedy, "Acute Insular Sclerosis and its concomitant Visual Disturbances,"*JAMA* 63 (1914): 2005. Kennedy answered by citing Uhtoff who said 50% but Kennedy wondered "if he does not mean that in half of the cases of disseminated sclerosis, is found temporal papillary pallor, " ibid., 2006.

122. Leo M. Crafts, "The Early Recognition of Multiple Sclerosis," *JAMA* 69 (1917): 1136.

123. Ibid., 1136.

124. Ibid., 1137.

125. Sachs and Friedman, "General Symptomatology of Multiple Sclerosis," 53.

126. Maurice Fremont-Smith, "On Certain Diagnostic Difficulties in Private Practice," *The Medical Clinics of North America* 10 (1927): 1317.

127. Ibid.

128. Ibid.,1325-26. The full case is as follows:

> well-developed and well-nourished woman of thirty-five, nervous and mentally depressed. She was of good color. Blood-pressure was systolic 110, diastolic 70. The pupils were equal and reacted to light; ocular motions were normal; the fundi were negative. There was no cranial nerve paralysis. The tongue was normal; teeth and throat, glands breasts, and thyroid negative. Lungs and heart were negative. The right kidney was palpable, abdomen was otherwise negative . . . The keen-jerks were equal and moderately active, ankle-jerks equal and normal. Biceps and triceps normal. There was no clonus and no Babinski. Vibratory and toe position sense were normal. There was not ataxia of legs. Romberg was negative. Sensation for pain and touch was normal. Blood Wasserman was negative. Red count, 4,650,000. White count, 6700. Differential, 83-12-3-2. The red cells were normal in size, shape, and color. Platelets were normal . . . Here again are we dealing with a nervous, poorly adjusted individual, with hysterical manifestations, or with a degenerative condition of the central nervous system? In favor of some sort of actual pathologic change were her previous robust health, her transient loss of vision five years before, the

temporary anesthesia of the face a year later, and the present numbness and tingling of her extremities. In favor of neurosis or hysteria were the glove-like distribution of her sensory symptoms ('numbness in both feet, less marked in legs up to knees, back and front'), the lack of confirmatory evidence on objective examination, the entire absence of objective evidence pointing to organic cord or blood disease, and last, her emotional reactions during examination, which were manifestly of the hysterical type. It seemed, moreover, logical that with definite symptoms appearing five years previously, some evidence of organic change should now be discoverable had these early symptoms been due to early actual disease. Arguing along these lines a diagnosis of hysteria was made . . . In January, 1926, nine months later, Dr. George Clymer found that her speech was scanning, 'she had a slight intention tremor, nystagmus, double Babinski, and suggestive though unsustained ankle clonus. I think there is no doubt at present that the diagnosis is multiple sclerosis.

129. Paul De Nicola, "Diagnosis and Treatment of Multiple Sclerosis," *NEJM* 209 (1933): 834-37.

130. Lewellys F. Barker, "Differential Diagnosis of Disseminated Sclerosis from other forms of multilocular encephalomyelopathy," *Bulletin Johns Hopkins Hospital* 58 (1936): 339.

131. George J. Wayne and William K. Bear, "Hysteria or Multiple Sclerosis," *Air Surgeon's Bulletin* 2 (1945): 234.

132. Ibid.

133. National Multiple Sclerosis Society, "Multiple Sclerosis Diagnosis and Treatment," *JAMA* 135 (1947): 569.

134. Richard M. Brickner, "Multiple Sclerosis," *Medical Clinics of North America* 32 (1948): 750.

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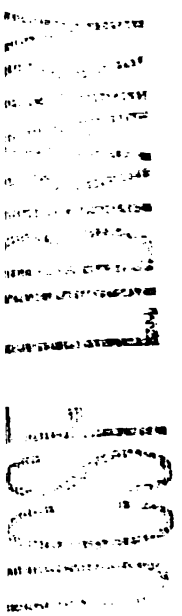
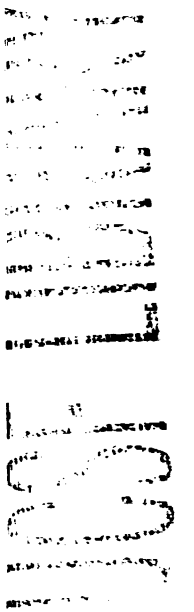
149. Kurland and Westland, 682-701.



Chapter Three
Vectors of Research on Multiple Sclerosis in the United States, 1870-1946: Boundaries in Question

I Introduction

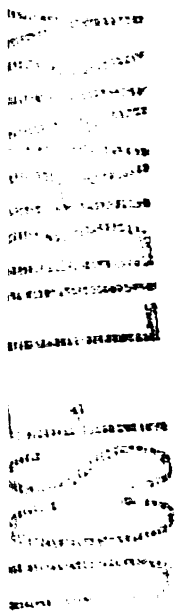
There never was a time through the 1950s when one particular etiological theory of multiple sclerosis dominated neurology. There were however recurrent theoretical themes concerning the etiology and pathogenesis of multiple sclerosis. Writers set the terms of the debate in theorizing and research on multiple sclerosis in the 1870s through the 1890s. Within this structure of theory, neurologists carried out laboratory and clinical work for seventy years. In this chapter I first lay out the basic theories about the causes of multiple sclerosis. I then explore specific research agendas in detail as the archival sources permit especially work on multiple sclerosis carried out at the New York Neurological Institute from 1920 to 1944 supported by the Commonwealth Fund. This is a useful case study for examining the consequences of a structure of medical research funding dominated by private foundations before World War II.[1] The analysis of research carried out at the New York Neurological Institute demonstrates how the structure of financing of research shaped the style of research. The autonomy of individual researchers led to a medical culture where the boundaries between the clinic and the laboratory and between experiment and therapy were quite fluid if they were distinguishable at all. Because of the broad range of topics considered appropriate for the still forming specialty of neurology, the boundaries between society and American neurology also remained fluid into the 1920s.



II Diathesis and Exciting Causes, 1870-1886

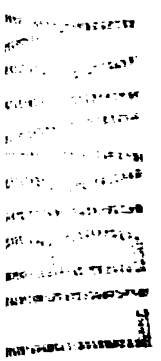
In the 1870s and 1880s writers expressed two basic opinions on the etiology of multiple sclerosis. One was that the cause was shrouded in mystery and virtually nothing

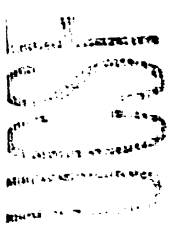
was known.[2] Other neurologists believed multiple sclerosis to be of "diathetic origin."[3] A diathesis was a "disease or taint which may be active at times, and which may be handed on to another generation."[4] In early and mid-nineteenth-century American medical thought, physicians thought that one inherited a predisposition toward manifesting some disease, what they termed a diathesis. During this time period in the United States many physicians and lay persons shared a set of assumptions about the diathetic origins of nervous disease.[5] Physician Jonathan Hutchinson explained in 1884 that a person with a diathetic weakness could "inherit the multiform varieties of scrofula, and among them abound the Protean forms of nervous disease, hysteria, chorea, neuralgia and epilepsy."[6] The concept of diathesis was part of a medical *mentalité* that saw nervous disease as the result of multiple influences including the weather, environmental conditions, lifestyle and habits, and constitutional inheritances.



In the 1870s and 1880s many authors posited that various exciting causes operating on a diathetic constitution could lead to multiple sclerosis; these included: trauma, fevers, exogenous toxins, overwork, heat stroke, exposure to cold, pregnancy, parturition, menstruation, emotional stress, metabolic disorder, or a primary infection.[7] This formulation of predisposing and exciting causes set the terms of debate for the next fifty years.[8] Within this etiological framework of predisposing and exciting causes American and European authors advances five basic theories of pathogenesis from the 1870s to the 1890s. One held that multiple sclerosis resulted from a derangement of the neuroglia, the result of a chronic inflammation or a developmental disorder. The second held that vascular disturbances caused multiple sclerosis. One variant of the vascular theory posited abnormal clotting leading to a thrombosis of venules; another variant held that the vascular problem was one of vasospasms leading to destruction of the myelin sheath. The third major theory was that multiple sclerosis resulted from infection either

by the direct action of an offending organism on the myelin sheath or the indirect action of a toxin caused by the presence of the infective agent. Researchers thought the most likely culprits to be first spirochetes then later filtrable viruses. The fourth important theory held that a metabolic disorder led to the production of endogenous toxins, either of sanguineous or lymphatic origin, which then caused multiple sclerosis. Finally a fifth and minor theory, in that it had few adherents, held that multiple sclerosis resulted from exogenous poisoning through exposure to lead, manganese, or other toxins.[9] Each of these pathophysiological theories had their advocates. Researchers in different times and places would take them up and abandon them. None of them was ever completely discredited or completely abandoned.[10] The question is why did particular neurologists take up particular theories in specific times and places? What were the factors which led a small group of neurologists or a single neurologist to choose to ask questions in a particular theoretical framework? Did American neurologists coordinate their research or did they work in a decentralized way?

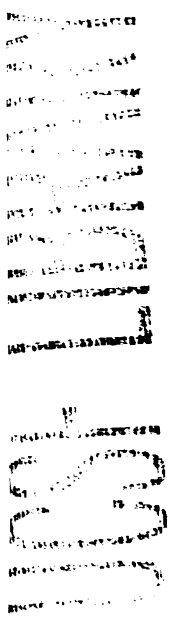




III Degenerationist/Hereditarian Theories 1887-1920

In the late 1880s neurological writers and researchers blended older notions of the diathetic origin of nervous diseases with theories of degeneration. This led to the idea that multiple sclerotics possessed a neuropathic constitution.[11] Many American neurologists continued to theorize that various exciting causes acted upon a neuropathic constitution leading to multiple sclerosis through the early 1920s. Then suddenly talk of a general neuropathic constitution dropped out of the medical literature. How does one explain the rise and fall of the neuropathic thesis in multiple sclerosis? Two main factors were most important: one was the social and cultural context of neurology in the United States and the other had to do with what research was possible for neurological workers from the late 1880s to the early 1920s in the United States.

The neuropathic thesis in the United States became conflated with degenerationist theories in the late nineteenth and early twentieth centuries. Degenerationist theories took on different meanings depending on their specific contexts. In Britain social critics tied degenerationist theories to questions of military preparedness and imperialism; in France public health officials linked degenerationist theories to fears about a declining birth-rate as compared with Germany; in Brazil physicians and social critics linked European degenerationist theories to anxieties about racial mixing; and in the late nineteenth and early twentieth-centuries in the United States, neurologists, physicians, eugenicists, and a broad spectrum of the educated middle and upper classes deployed degenerationist theories to explain the social stresses caused by urbanization and mass immigration.[12]



In the late 1880s the impact of degenerationist thought on constructions of multiple sclerosis first began to appear. This thinking covered most nervous diseases and had antecedents in the diathetic theories of the mid-nineteenth century. In 1887, George T. Stevens, a professor at the Albany Medical College, wrote that "a very large portion" of nervous diseases were hereditary. Nervous diseases were "among the group of disorders which, through hereditary tendency, may manifest themselves either in the same manner or interchangeably."[13] Stevens thought that the importance of predisposition in nervous diseases had not been given adequate attention by previous neurologists.[14]

In the late 1880s and early 1890s American neurologists would begin thinking about nervous diseases in hereditarian terms. James Jackson Putnam reported in 1891 that there was a "neuropathic inheritance" in some of his multiple sclerosis cases.[15] In 1892 Charles L. Dana, Professor of Neurology at the New York Post-Graduate Medical School, taught that multiple sclerosis was a chronic degenerative disease and that

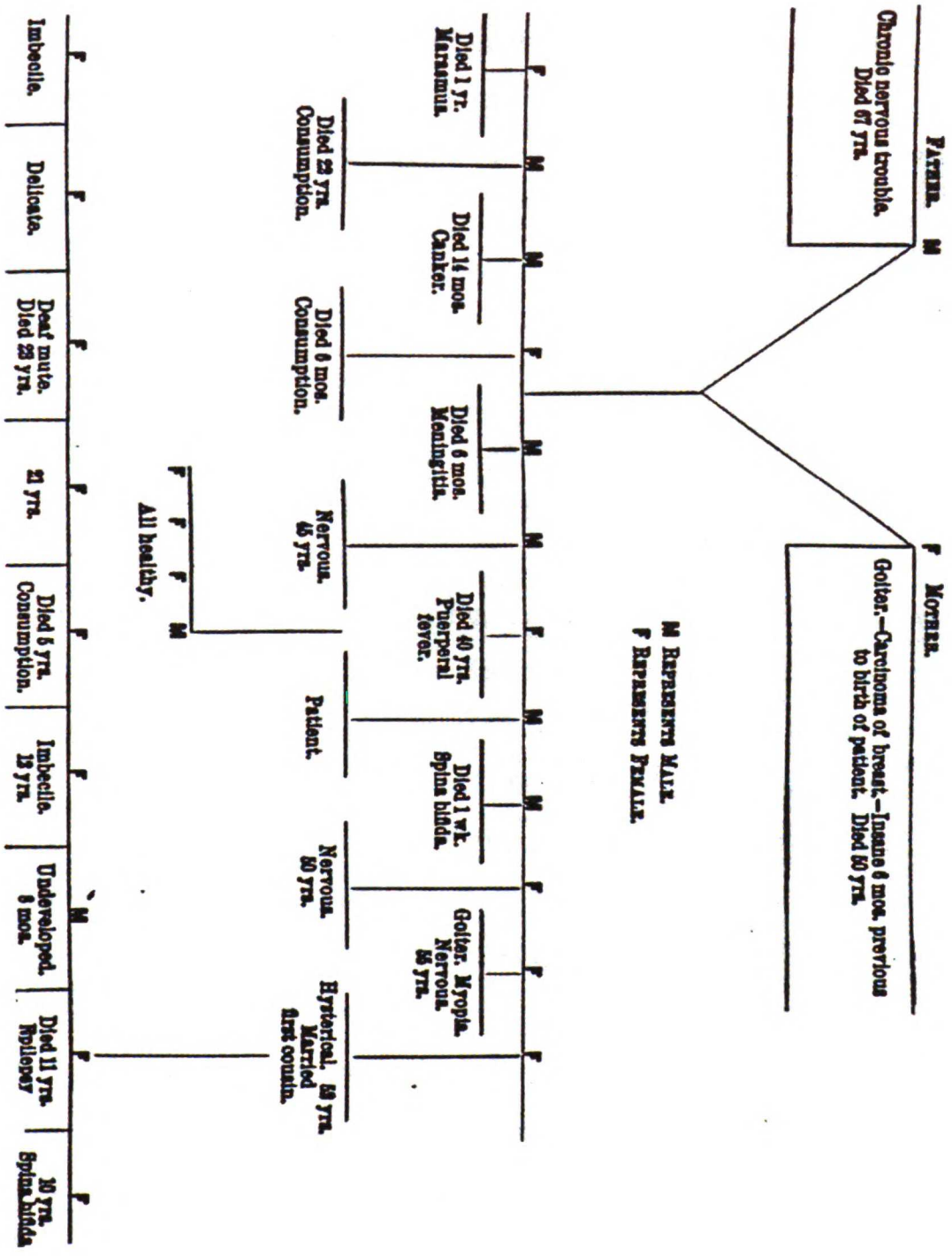
"generally the patient inherits a neuropathic constitution."[16] Dana concluded that, in fine, acute infections and severe traumatisms involving concussion, co-operating with a neuropathic nervous system, form the most important etiological factors."[17] In a presentation before the Association of Assistant Physicians of Hospitals for the Insane, in Cleveland, Ohio, September 26 to 28, 1899, Drs. Irwin H. Neff, Assistant Physician, Eastern Michigan Asylum, and Theophil Klingmann, Pathologist to the Michigan Asylums, suggested that 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.[18] In front of the New York Neurological Society, in 1905, Dr. I. Abrahamson presented two brothers, aged 19 and 15, diagnosed with multiple sclerosis, who upon examination were found to have "many stigmata of degeneracy."[19] Figure 1 shows this style of reasoning in graphical form. The "patient" is a multiple sclerotic.[20]

For epilepsy, similar sorts of arguments were put forward. Epileptics also had the "stigmata of degeneration" and neurologists constructed elaborate family trees with reference to alcoholic, syphilitic, and tubercular relatives indicating a "defective taint."[21] In *Heredity in Relation to Genetics* (1911), Charles Davenport argued that hemophilia, otosclerosis, Huntington's chorea, insanity, epilepsy, alcoholism, pauperism, criminality, feeblemindedness, and multiple sclerosis were heritable.[22]

William A. White, Superintendent of the Government Hspital for the Insane, Washington D.C. and Professor of Nervous and Mental Diseases at Georgetown University and George Washington University, in a 1913 neurological textbook article entitled, "Eugenics and Heredity in Nervous and Mental Diseases," synthesized Mendelian genetics, neurological thought, and eugenics to explain many nervous diseases. White wrote that his project was concerned with race betterment. He warned of

Figure 1

Tainted Heredity Chart (1899); Patient = Multiple Sclerotic



the economic drain of the "great army of the unfit." He classified the epileptic, the criminal, the deaf, dumb, blind, crippled, and paupers as among the classes of the unfit.[23] White taught that a neuropathic constitution was inherited. Some diseases like paralysis agitans, Huntington's chorea, and multiple sclerosis developed later in life and were more difficult to place in Mendelian perspective. However, White maintained that "by constructing elaborate family trees reaching back through several generations that it may not infrequently be possible to trace a bad strain and see its culmination in certain individuals."[24] He displayed a pedigree chart indicating that a predisposition to multiple sclerosis was perhaps a recessive trait since instances of direct inheritance were infrequent.[25] Figure 2 shows a brother and sister with multiple sclerosis.[26]

In World War I, partly through statistics garnered during massive medical examinations of drafted soldiers, a geographic pattern to multiple sclerosis emerged. The northern latitudes of the United States had much higher proportions of multiple sclerosis than did more the southerly regions. This mirrored European epidemiological data that had found MS to be more prevalent in the northern areas of Great Britain and in northern Europe, especially Scandinavia. In terms of ethnicity in the United States, the study displayed charts which claimed that people of English, Scottish, German, Swedish, Norwegian, and Finnish stock had a much higher incidence of multiple sclerosis than did people whose ancestors were from eastern and southern Europe. In terms of race, African-Americans, Asians, and American Indians had the lowest incidence. Commenting on this perception of MS one commentator wrote that "strangely, blond, blue-eyed people seem to be most susceptible."[27] In Figure 3, the authors wrote Scandinavians near the northern states with a higher prevalence of MS.[28]

In the 1950s this ethnic framing disappeared. This ethnic and racial way of framing MS in the 1910s took these social categories as unproblematically natural as if

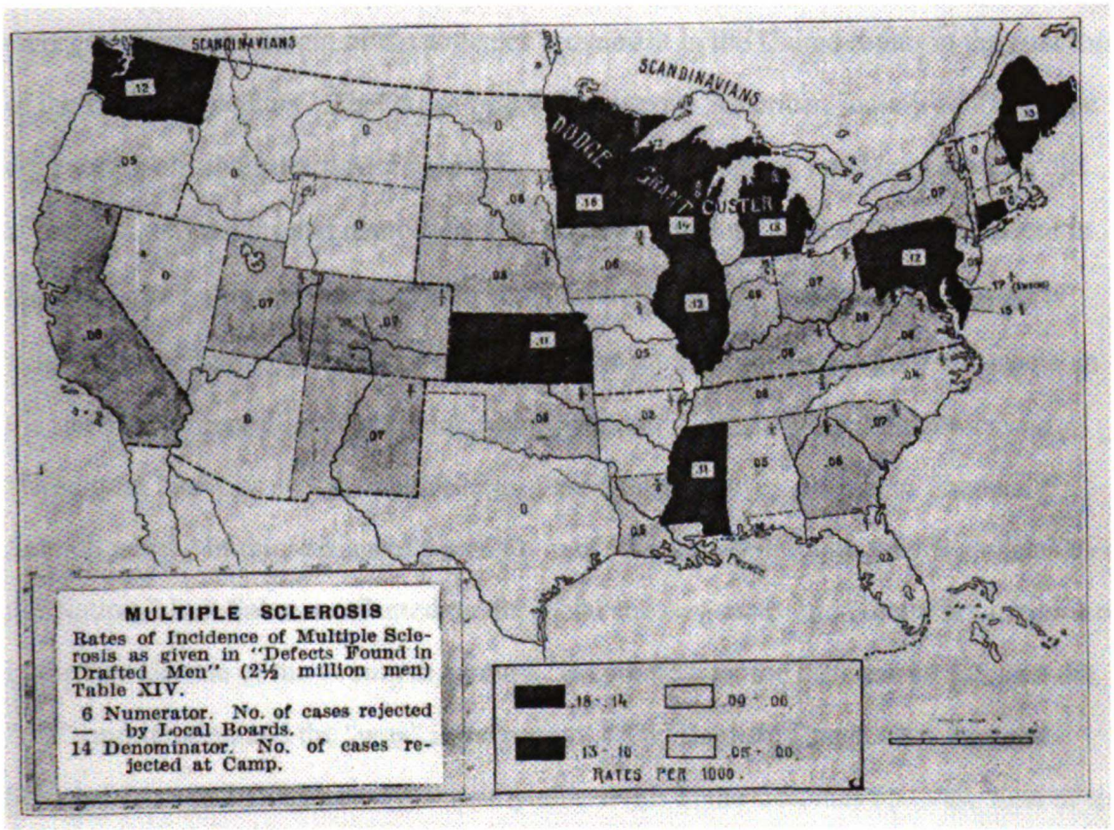
Figure 2

A Brother and Sister with Multiple Sclerosis (1909)[1]

[1] T.H. Weisenburg, "Multiple Sclerosis; its occurrence in families: brother and sister," *Archives of Diagnosis* 2 (1909): 167-74.

Figure 3

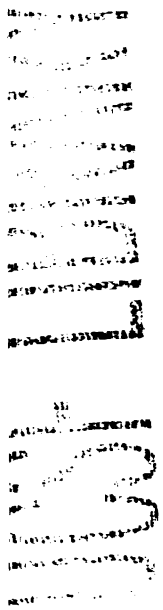
Rates of Incidence of Multiple Sclerosis (1917-18)-Defects in Drafted Men-Scandinavian[1]



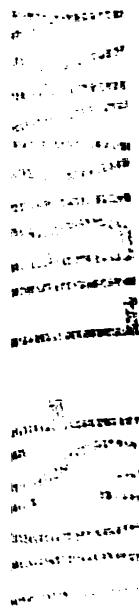
[1] Association for Research in Nervous and Mental Diseases, *Multiple Sclerosis* (New York: Paul B. Hoeber, 1922), 9.

ethnic and racial groups did not intermarry or at least have sex with one another creating children of mixed heritage. It was not that no people coded African-American had MS or that no Italians had MS. The researchers considered the occurrence of MS in these groups insignificant "noise" in the data. This was not an inevitable framing choice but one which was powerfully embedded in the social and neurological thinking of the day.

This construction of the ethnic and racial distribution of multiple sclerosis emerged during the height of the eugenics movement in the United States in the first third of the twentieth century. The"broad middle" of literate American society conflated the degenerationist theories of Morel, Nordau, Lombroso, Magnan, and Hammond, as did zoologists, physicians and neurologists.[29] Eugenic and neurological thought existed in the same discursive and social spaces, that is simultaneously within the profession of neurology and within the non-neurological native white middle and upper classes.[30] In the same 1913 neurological textbook where William A. White wrote "Eugenics and Heredity in Nervous and Mental Diseases," mentioned above, physician Thomas W. Salmon wrote "Immigration and the Mixture of Races in Relation to the Mental Health of the Nation." In it Salmon reflected on the social stresses occasioned by mass immigration in the first decade of the twentieth century. He pointed out the differences between the "old" immigration and the "new" immigration. "The English, Dutch, Germans and Scotch-Irish which constituted practically all arrivals in the Colonies up to the time of the Revolutionary War were closely akin and, centuries earlier, they had been one Germanic race in the forests surrounding the North Sea."[31] As for the German and Irish immigrants of the mid-nineteenth century, he asserted that, "there can be no doubt that the assimilation of Germans has been complete and satisfactory."[32] The Irish had been assimilated, although with a little more difficulty.[33] For Salmon it was self-evident that these earlier immigrants "constituted the best racial stocks in Europe."



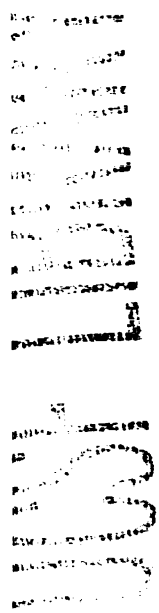
The Italians, Slavs, and Jews together with smaller groups from southern and eastern Europe were characterized as the "new immigrants."[34] Salmon maintained that these new immigrants could not be easily assimilated and that one result of this was the "permanent exile in this country of large numbers of the insane and those ill from other causes," who, after becoming ill in the United States, were denied re-entry to their countries of origin.[35] These new immigrants were seen as poorer than the "old" immigrants and forced to huddle in crowded cities unlike their predecessors. This led to a higher prevalence of nervous diseases among the new immigrants which was to be expected since Salmon and his colleagues "knew" that "the influence of race upon the susceptibility to disease is very great."[36] They saw Italians as having the highest rates of syphilis and general paresis of any group in the country because their group was composed of so many single men and married men living apart from their wives. The Japanese had a general attitude toward self-destruction. There was a "strong tendency to delusionary trends of a persecutory nature in West Indian negroes." The Hebrews had "hidden sexual complexes" and had higher incidence of "manic-depressive psychosis, dementia praecox, the psychoneuroses, and psychoses constituted with constitutional inferiority." The Poles had a "remarkable prevalence of mutism" and Slavs in general were more likely to have alcoholic psychoses and were twice as likely to have general paresis as the native-born population.[37] Salmon worried that there would be a "substitution of Slavs, Italians, and Hebrews for native racial stocks." This of course would lead to higher levels of "insanity, mental defect, and organic nervous diseases."[38]



Some patients at least shared the neuropathic taint theory. This is the voice of a patient whom neurologists diagnosed as multiply sclerotic in 1915. In his diary entry of December 5, 1915 W.N.P. Barbellion wrote: "Spent the last two days, both of us, in a state of unrelieved gloom. The Clouds never lifted for a moment-it's awful. I scarcely

have spoken a word . . . And eugenically, what kind of an infant would even a Mark Tapley expect of a father with a medical history like mine, and a mother with a nervous system like hers? . . . Could anything be more unfortunate?"[39]

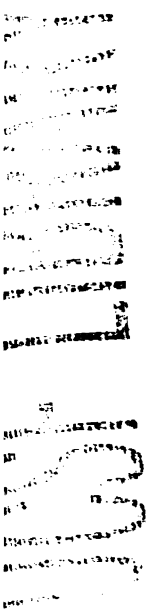
This framing of MS was, therefore, not a unique formulation but one that covered nervous afflictions generally. It was bound up with neurological thought which was embedded in the general cultural anxieties of the day which mass immigration had inflamed. Neurologists wrote simultaneously as professionals and citizens in neurological textbooks and in popular journals. This was partly because neurology as a specialty was still looking for its own voice in the American context which would differentiate itself from psychiatry and psychology. Ironically, as neurology became more "scientific" it became less powerful and held in lower esteem than psychiatry which won the battle for which specialty group could claim expertise over a medicalized society. Neurology seemed less relevant to society and an area of medicine which appeared hopeless. As Dr. Bernard Glueck commented in 1927, the "neurological school . . . deals with hopeless material from beginning to end."[40]



Neurological, eugenic, racial, and social thinking occurred in the same discursive space and often by the same authors. Thinking about multiple sclerosis was entangled with racial and ethnic thinking in the context of American neurological practice which partly explains why the neuropathic taint theory of multiple sclerosis held sway from the late 1880s to the early 1920s. Another reason was that before the 1920s, the possibilities for laboratory and physiological research in neurology in the United States were quite limited. Creating elaborate family trees, together with clinical diagnosis, and the translation of European neurological works, were the activities which constituted neurological research in the American context. Why then, did the neuropathic taint theory decline in the medical literature after the mid-1920s? One reason is that neurologists abandoned the neuropathic theory as they abandoned eugenics in the 1920s.

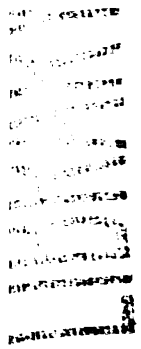
Another reason is that American neurologists were attempting to give their poorly esteemed specialty on stronger structural, intellectual, and clinical foundations. Since therapy followed etiological theory in a medical culture dominated by the doctrine of specific etiology and the germ theory of disease, what good would a theory of neuropathic taint do for the clinician for whom patient consultation was the economic basis of his work?

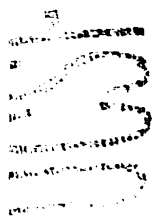
This general question came up at a 1924 conference of the Association for Research in Nervous and Mental Diseases (ARNMD) devoted to "Heredity in Nervous and Mental Disease."[41] Neurologist Smith Ely Jelliffe presented a paper in which his main point was that the phylogenetically and ontogenetically younger parts of the nervous system, specifically the pyramidal tracts, were more likely to degenerate first. After the paper neurologist Charles L. Dana asked, "I have always been interested in the preventive side of medicine, and these diseases about which we are talking now, and those particularly which Dr. Jelliffe has referred to, are pretty nearly altogether what we call 'dead stuff'; we can do nothing for them except study them genetically and clinically. Is there anything in your method, which would encourage preventive medicine as regards these maladies?" Jelliffe responded, "Yes, I have in many instances, in my paper, referred to the eugenic aspect of the situation."[42] Obviously this held little promise for the clinic. This is partly why three years earlier at a conference on MS The Commission of the ARNMD, designed to "sit and hear the evidence," concluded that while there was often evidence of a neuropathic taint in multiple sclerosis cases the most promising approach lay in bacteriology.[43] Hereditarian/Degenerationist ideas declined in neurology (and in social science thinking) in the late 1920s as other ideas increased in importance. If multiple sclerosis could be found to have a single cause as had recently been demonstrated in syphilis and neurologists could deploy a specific therapy such as



Salvarsan, the prestige of neurology would increase and their specialty would have a firmer basis in practice.[44] This would also place research on MS more clearly within the doctrine of specific etiology and make neurology more medically modern.

Not only did the bacteriological approach offer more possibilities for immediate financial and professional gain, new neurological tools made the posing of different questions possible. Before the 1920s the neuropathic hereditarian vector in research was partly determined by the research tools available to American neurologists. In other words, it was one of the few activities that could be deemed research which Americans could actually do given the structural and institutional limitations they faced. By the 1920s new tools such as the lumbar puncture and biochemical methods for studying the spinal fluid and blood made new lines of inquiry possible.[45] These new tools arrived along with an increase in medical research generally made possible by the philanthropy of private foundations in the United States.



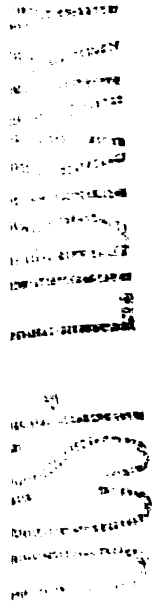


IV The New York Neurological Institute and the Commonwealth Fund: The Structure of Funding and Research on Multiple Sclerosis, 1919-1944

During the 1920s through the 1940s the Commonwealth Fund supported studies on multiple sclerosis at the New York Neurological Institute which became the Neurological Institute at Columbia University. An analysis of this funding source and the laboratory research it supported revealed how the interests of a specific medical institution, the specialty of neurology, and a private foundation came together in a program of research in multiple sclerosis. Beginning in 1920 the Commonwealth Fund appropriated five thousand dollars a year for research on multiple sclerosis at the New York Neurological Institute (NYNI).[46] They did so because neurology seemed "to be one of the fields of medicine and public health" which was "as yet relatively unoccupied, especially by the other foundations." The Rockefeller Foundation had encouraged the

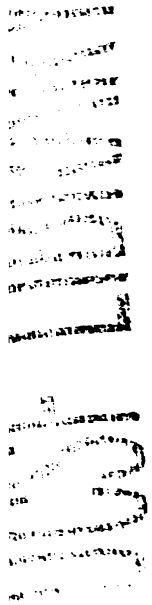
formation of "psychiatric institutes" but there were no other institutes in the United States devoted solely to neurology. The Commonwealth Fund perceived that the general medical experience of World War I brought neurology "prominently to the fore" as an underdeveloped area of medicine. Also, during the war the Surgeon General had assigned physicians to the New York Neurological Institute for training which the Commonwealth Fund believed showed the need for such an institution.[47]

Until 1919 the NYNI concerned itself mainly with direct patient care and education. Since its opening in October 1909 the NYNI had received 16,000 patients in its wards and 45,000 in its dispensary, and physicians gave "upwards of 300,000 treatments."[48] Because NYNI physicians concerned themselves mostly with patient care, especially the dispensing of Salvarsan for syphilis of the central nervous system, and education, they had little time or money for research. The Commonwealth Fund noted that there was a "keen and growing appreciation of the necessity of research" in neurology. The problems they saw as most pressing after conversation with neurologists were epilepsy, multiple sclerosis, sleeping sickness, and poliomyelitis.[49] The Commonwealth Fund committed itself to funding research at the NYNI to advance knowledge in an area of medicine that was underdeveloped in the United States and for which they perceived a substantial need.[50]



In 1919 neurology had still only partly emerged as a specialty distinct from general medicine or psychiatry unlike in Europe.[51] At that time the boundaries between what we would consider psychiatry, neurology, and psychology were fluid as health workers constructed their respective specialties. Neurologists claimed certain diseases for their own as part of construction of the foundation of their specialty. Multiple sclerosis was a key disease on which neurologists made a unique claim on the professional geography of the body.

Neurology's interest as a specialty intersected with the Commonwealth Fund's interest as a pragmatic charity. A 1920 report to the Director's of the Commonwealth Fund stated that it was "probable that a large number of diseases of the nervous system will be found to be preventable or curable in the future, especially diseases such as epilepsy, apoplexy, multiple sclerosis, locomotor ataxia, functional affections and many new growths of the central and peripheral nervous system. It is questionable whether these may be regarded as purely nervous diseases, but some of them are recognized generally by physicians as being primarily in the field and they afford and excellent opportunity for research."[52] The two diseases the NYNI neurologists chose to establish a research foundation for their specialty and their institution were multiple sclerosis and epilepsy.[53] The Directors of the Commonwealth Fund found multiple sclerosis research project to "be exactly of the kind which the Directors have in mind in offering support for the extension of medical research" because they hoped to obtain quick results and to solve a problem which had a significant "economic impact" on the country.[54] Multiple sclerosis met the needs of the philanthropic entrepreneuralism of the Commonwealth Fund and the institution needs of the New York Neurological Institute in particular and American neurology in general. The disease category served to "rationalize, mediate, and legitimate" the relationship between the Commonwealth Fund and the New York Neurological Institute and became the basis for their ongoing negotiations of sometimes conflicting goals.[55]



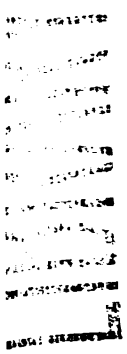
In the advent of foundation support for neurological research one sees a theme which will recur for the next forty years; that is, neurologists used research dollars designated for specific diseases to support research they were already engaged with, to strengthen their local institutions, and to construct the boundaries of their still only partly formed specialty; however, the Commonwealth Fund wanted to finance neurological

projects which might quickly result in practical application such as disease prevention, therapy, and diagnostic tests. These differing goals made for a sometimes strained relationship between the Commonwealth Fund and multiple sclerosis researchers at the NYNI from 1920 to 1934. The Commonwealth Fund wanted a product they could point to and claim philanthropic success and the NYNI wanted money for an ongoing research program which would advance their individual careers, lead to journal publications, and allow them to build up the laboratory facilities of their own institution, specifically animal and biochemistry labs.[56] The key issues of conflict between the researchers and the Commonwealth Fund officers which emerged were who would decide which projects were to be funded? Who would evaluate the ongoing work? Who would decide in which direction research could go? How were different research programs to be coordinated? Why were there differences between research proposal representation and actual research practices?

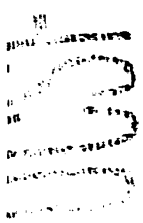
In January 1921 lead neurologist Frederick Tilney outlined three main lines of inquiry into multiple sclerosis: bacteriological, biochemical, and developmental pathology. Remembering from the first section these three areas of neurological speculation had been around since the late nineteenth century; that is, looking for a specific infectious agent, looking for an endogenous lipolytic substance, and looking for developmental abnormalities of the neuroglia. The ideas were not knew. What was new was the possibility of building laboratory facilities and relatively large amounts of money to make an ongoing research program possible. Three different researchers eventually went off in these directions at the New York Neurological Institute. Dr. Oscar Teague began looking for a possible infectious agent as the cause of multiple sclerosis.[57] Leon Cornwall eventually replaced Teague. Frederick Tilney studied the development of myelin in rats. Richard Brickner searched for a lipolytic substance in the blood which might attack myelin. Analysis of these projects reveals something about how the

structure of foundation financing affected the direction of multiple sclerosis research and the conditions of laboratory work on multiple sclerosis.

Basically this particular model of foundation funding led to laboratory practices at the NYNI characterized by high autonomy for the individual researcher with little oversight or coordination with other researchers. For example, even before Teague completed his study, Tilney wanted permission from the Commonwealth Fund to study epidemic encephalitis "in the event that this lead in multiple sclerosis proves unproductive." The Commonwealth Fund refused permission for Teague to study epidemic encephalitis, preferring instead that he study sleeping sickness. Nevertheless, Teague's replacement in April 1922, Leon Cornwall, ignored the Commonwealth Fund request and studied epidemic encephalitis while at the same time continuing to study multiple sclerosis. This was the direction Cornwall wanted to go; so he did. The only option the Commonwealth Fund had was to withdraw funding.

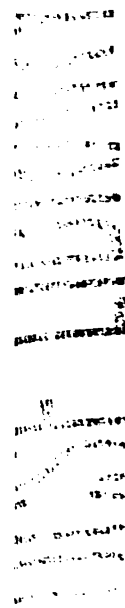


Likewise, Frederick Tilney used his Commonwealth Fund money to study the development of myelin in rats directed toward understanding multiple sclerosis. However he diverted a large portion of his resources to coordinating myelin studies with the psychiatric questions of delinquency, degeneracy, and mental defectiveness. Tilney wrote in 1929 that "the specific problem involved in Multiple Sclerosis opens directly upon the broad and extremely important one of human behavior. . . . Idiots, morons and imbeciles show a smaller degree of myelinization in their brains . . ."[58] Tilney had used his money to study the psychiatric questions in which he was already interested. As the previous section has shown, Tilney's work was rooted in a tradition of neurological thought which was decades-old by the 1920s. Indeed Tilney was a senior neurologist and of a different generation than Brickner and Cornwall.



In 1927 the Commonwealth Fund contemplated withdrawing funding because the Neurological Institute researchers were using their appropriations for purposes not in

their original proposal. Barbara S. Quinn , Assistant director of the Commonwealth Fund, worried that the NYNI researchers were not making a "concentrated drive" in a "productive direction." The Commonwealth Fund wanted demonstrable results in either prevention, diagnosis, or treatment. Instead they perceived that they were "entering upon an indefinite period of subsidizing researches on problems so difficult that they may consume much of the energy of the next medical generation."[59] Dr. M.C. Winternitz in correspondence with Quinn wrote that after reading their initial prospectus and report he was "left with the distinct impression that the funds placed at the disposal of these physicians have been utilized for the general conduct of their investigative work, irrespective of the programs they have outlined. One assumes . . . that the funds made available by the Commonwealth Fund are the major resources for the investigative work being conducted by Dr. Tilney and Dr. Elsberg <working on epilepsy> and their co-workers, and the corollary of course is that they do not receive any considerable budget from other sources. I mention this because it seems to me that you have voted money for very specific investigations, and irrespective of the value of the work that has been done in the past, one would have to admit that only a relatively small portion deals with the outlined problems. . ."[60]



The problem was how to evaluate the technical progress of the studies in mid-stream. The initial funding had not gone through a peer-review process. This innovation would not develop in MS funding until the late 1940s. Winternitz warned Quinn that because "Dr. Tilney and R. Elsberg occupy positions in the front rank of American neurologists" it would be difficult to get objective opinions on their work from other neurologists.[61] This was especially true because "the difficulties are always very great in getting really conscientious judgments from such people about the work of others in similar fields, especially where financial considerations in support of such work are in the

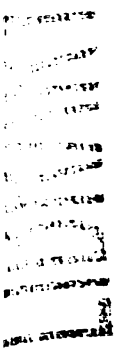
balance."[62] Quinn discovered that Winternitz was correct writing that "the general attitude toward anything which Dr. Tilney and Dr. Elsberg may do is certainly true and is necessarily handicapping me in my attempt to secure expert technical opinion."[63] The Commonwealth Fund sent letters asking technical opinions on continuing Tilney's MS funding and Elsberg's epilepsy funding to: Dr. Harvey Cushing, Peter Bent Brigham Hospital, Boston Massachusetts; Dr. Hugh Talbot Patrick, Chicago, Illinois; Dr. Theodore Weisenburg, Editor in Chief of the *Archives of Neurology and Psychiatry*, Philadelphia, Pennsylvania; and Dr. Albert Barrett, State Psychopathic Hospital, Ann Arbor, Michigan.[64]

Their responses show that what counted in evaluating research was not the merit of the proposal but the reputation of the individual researcher. Albert Barrett endorsed the proposals saying that "those who are in charge of the guidance of these researches are so well known for their ability and research interests that I sincerely hope that the Commonwealth Fund will continue to give their support to their work."[65] Hugh T. Patrick responded that while he was "not familiar with all the details of the research work you mention . . . I am very certain that it is valuable work. Anything that Tilney does is worth while. To be entirely frank with you, I might add that Dr. Elsberg has not as high an order of intellect as has Dr. Tilney and consequently his own original work is less valuable but Dr. Elsberg's industry and energy are wholly admirable. In other words, I believe that withdrawal of the Commonwealth Funds' support for the research of these two men would be entirely regrettable."[66] Even in the negative, what counted was reputation not the particulars of a proposed study.

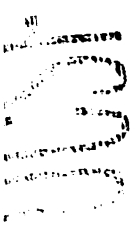
In the report to the Commonwealth Fund Board of Directors, it was the reputation of Tilney that Quinn emphasized. She also pointed out that the fund had appropriated five thousand dollars a year for multiple sclerosis research since 1920 giving the sense

that they were too far along to back out now. If they did so there would not be any clear product of their philanthropic entrepreneuralism whether in disease prevention, diagnosis, or treatment.[67] In 1928 the Commonwealth Fund decided to continue funding research on multiple sclerosis through early 1934.[68]

This continued funding allowed Leon Cornwall and Richard Brickner in 1928 and 1929 to begin investigating whether there was some substance in the blood or spinal fluid of multiple sclerosis patients which destroyed myelin.[69] Though Tilney represented himself as being in close contact with the work, by 1934 the Commonwealth Fund had concluded that Dr. Tilney was "not keeping in closest touch with the research . . . It is evident that Dr. Tilney has not checked carefully, as he could give no details whatever" on Cornwall's and Brickner's work.[70] Indeed, as the end of the funding approached in 1934 it became apparent that the representations of a unified and collaborative attack on multiple sclerosis at the NYNI as found in letters and reports to the Commonwealth Fund were mostly fictions.



In this example one sees how in practice the researchers went their own way with little supervision or coordination. By 1931 Richard Brickner believed he had found "an abnormal lipase (fat dissolving ferment)" in the blood of multiple sclerosis patients. He had begun to use quinine as a treatment because he thought that "that the abnormal ferment in the blood can be controlled by the administration of quinine."[71] Brickner began giving quinine to multiple sclerosis patients and obtained what he thought were favorable results.[72] In 1934 Brickner presented his quinine research to the "Neurological Section of the New York Academy of Medicine and was there criticized for failure to test the method of therapy by comparison of treated cases with an untreated control group."[73] As it turns out the person who made the criticism at the meeting was Leon Cornwall, also of the New York Neurological Institute, also working on multiple sclerosis. In a candid



1934 interview with a Commonwealth Fund official Cornwall related to a Commonwealth Fund interviewer that "the research at the Neurological Institute is not correlated between the different workers and that obviously Dr. Tilney is giving relatively little attention to it. For example, Dr. Cornwall has not followed Dr. Brickner's work and had remained relatively ignorant of the results until it was reported a few days ago at the New York Academy of Medicine. At that time Dr. Cornwall immediately recognized some of the weakness of Dr. Brickner's research and it was he who pointed out in a meeting that the quinine therapy studies ought to be subject to a control series of cases."[74]

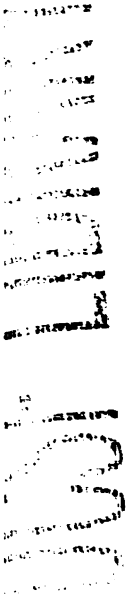
Tilney had been the point man in the negotiations with the Commonwealth Fund and had repeatedly promised that a therapy, a diagnostic test, or a significant discovery was just around the corner. By 1934 it became clear this was not the case. In the same interview Cornwall took "a much less optimistic <sic> attitude to the results of the study at the Neurological Institute than" had "Dr. Tilney, and" was "rather inclined to believe that still comparatively little is known about the disease. . . . as regards the fact that the destructive process is caused by a ferment in the blood stream while undoubtedly significant is in Dr. Cornwall's opinion not entirely clear cut as the same or very similar ferment can be found in non-neurologic diseases . . ."[75] The Commonwealth Fund declined to continue funding in 1935 believing that the researchers at the New York Neurological Institute had reached a dead end.[76]

This pattern of granting funds based on the reputation of the researcher rather than through peer review and the subsequent channelling of those funds to previous interests happened again when the Commonwealth Fund appropriated money to the New York Neurological Institute during World War II to meet a financial crisis caused by the conflict. In order to better understand this later episode one needs some basic

information about developments at the NYNI. The New York Neurological Institute hired Tracy Jackson Putnam as its director of the neurology and neurosurgery services in 1939. Columbia University appointed him professor in both fields simultaneously.[77] Putnam had been Professor of Neurology at Harvard University and Chief of Neurological Unit at Boston City Hospital. In 1940 he became editor of the *Archives of Neurology and Psychiatry*.[78] Through 1939 Putnam had published one hundred articles on neurology and neurosurgery.[79] Since 1925 the New York Neurological Institute had become officially associated with Columbia University and in 1937 became associated with Presbyterian Hospital. The financial arrangements for research thus became more complicated. As Tracy Putnam put it in 1941: the "research activities of the Neurological Institute and the staff of the Department of Neurology . . . are so intertwined, in their professional activities and in the various budgets, that it is almost impossible to unscramble them."[80] Neurological research received funding from the university, from a Presbyterian Hospital gift, and the Rockefeller Fund. From these multiple sources Putnam created a single pot from which to fund research and clinical activities. Unfortunately the war meant that the funds were not sufficient and the Institute was running a "huge deficit."[81] Therefore the Commonwealth Fund granted eight thousand dollars a year for 1941/42, 1942/43, and 1943/44.[82]

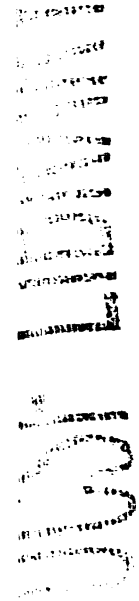
The Commonwealth Fund appropriated money for a three year study of multiple sclerosis directed by Putnam. The study was based on Putnam's work conducted in Boston before he came to New York where he theorized that a thrombosis of venules was the precipitating physiological event which led to demyelinization. He began testing dicoumarin, an anticoagulant, at the New York Neurological Institute as a preventive therapy for MS. In the foundation reports of Putnam's work the emphasis is on Putnam's character not the quality of the work being performed. A 1943 Commonwealth Fund

memorandum reported that Putnam's work "has been done or is well underway exactly in accordance with the statement made in the Board report of April, 1942."[83] What mattered was that Putnam was living up to his gentlemen's agreement unlike his predecessor Tilney. However Putnam was using the Commonwealth money to support a previous interest of his, i.e. his thrombotic etiology theory, just like Tilney had used Commonwealth money toward his psychobiological interests. A 1944 memorandum by the foundation officer Dr. Heffron, after uncritically summarizing the work being conducted by Putnam, said that "the more I get to know Putnam the greater respect I have for his ability to secure excellent workers and to get at the heart of extremely complicated and difficult problems."[84]



As it turns out the situation with Putnam and his workers was not as rosy as the positivist reports of the Commonwealth Fund portrayed. In November 1945 the lay president of the Neurological Institute Board, a Mr. Cooper, asked Putnam to resign. This request was not supported by Dean Willard C. Rappleye of the College of Physicians and Surgeons. Nevertheless, it does reveal that there was significant dissension among the neurologists working under Putnam. Dean Rappleye gave an indication about the basis for the conflict; when interviewed he said: "it has been evident for several years that P. <Putnam> was trying to do too much--teaching, research, direction of the clinical service, and a heavy operating schedule. The result is that administration has suffered. When P. <Putnam> came to New York it was not expected that he would do neurosurgery. Apparently the need of increased income to meet a domestic situation led P. to go in heavily for operative work. This has naturally caused some feeling among local neurosurgeons. R. <Rappleye> advised P. <Putnam> long ago to give up the administrative side and put all his energies into teaching and research."[85] Later Rappleye concluded that Putnam had "seriously neglected the administration of the

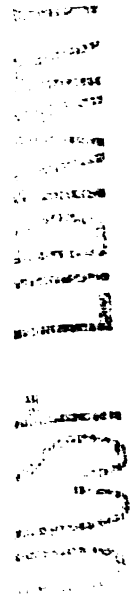
unit, failing even to attend any teaching rounds or conferences."[86] Putnam himself had a different story. He claimed that when he was hired he was not expected to do administrative work, that he was permitted to have a private practice, and that he was to have a free hand in directing the neurological and neurosurgical services. He further claimed that the Presbyterian Hospital was attempting to take over the Neurological Institute and turn it into a hospital department with a focus on clinical activities only. He added that hospital and university administrators were interfering in his direction of the Institute.[87] Later Putnam claimed that he had been asked to fire all the Jews in the department and he had refused to do so. There were many refugee physicians working in the Neurological Institute during the war. It may have been that he was asked to sack these physicians. However, it should be noted that Columbia University had a well-known history of anti-Semitism. At any rate this episode revealed the messy institutional realities within which research was carried out. In reading the reports of the Commonwealth Fund one is struck by the linear and positivistic descriptions of research presented to the Fund Directors. The Directors and often the foundation officers believed they were funding "pure" research leading to "advances" in knowledge. The reports glossed over the complexity of the questions involved and the extreme difficulties encountered in baffling neurological diseases. These representations, though elegant, were a distortion of the actual conditions of laboratory research at the New York Neurological Institute. They made promises of progress which fit into the general cultural narrative of medical progress but which were likely to lead to disappointment given the difficulty of the diseases being studied.



Nevertheless, this style of funding research, that is a gentlemen's agreement between the Commonwealth Fund officer and the lead neurologist, in this study, Tilney or Putnam, led to an individualistic style of research. A consequence of this style of research meant that the boundaries between the laboratory and the clinic and between

practices considered therapeutic and those considered experimental would be blurry if distinguishable at all. The reason was that once funded the individual researcher had the power to decide to decide the direction research should go in, how to conduct the experiment in question, and what was a proper therapeutic course for patients. Easy distinctions between researcher and clinician and the laboratory and the clinic are unsustainable during this period.

There was a continuous material and semiotic loop between the clinic where researcher/physicians saw patients and the laboratory where these same workers studied the blood and spinal fluid of MS patients. As one Commonwealth Fund officer discovered in an interview with Tracy Putnam: "all in all, the work supported by our appropriation is partly research and partly service. At times the two are so completely intermixed that it is highly artifical <sic> to attempt to separate them."[88] This laboratory research work was done with a clearly clinical goal in mind whether in diagnosis or treatment. For example, Oscar Teague and later Leon Cornwall attempted to find an infectious organism causing MS from 1920 through 1926. Even though Cornwall had negative results in terms of isolating a pathogenic organism he gave "arsenic preparations that were known to affect favorably other diseases due to spirochetes" to "cases of Multiple Sclerosis with negative results."[89] At another Manhattan institution, the New York Hospital, physicians gave Salvarsan to multiple sclerosis patients as well during this time period.[90] This was not out of the mainstream of neurological thought in that the consensus of the Commission of the Association for Research in Nervous and Mental Diseases had thought the bacteriological approach the most promising in 1921. The point is that there was a fluid boundary between the clinic and the laboratory and neurologists viewed the clinic as an extension of the laboratory.



In another example, when Richard Brickner believed he had found a lipolytic

enzyme he began to treat patients in his clinic with quinine. There was no peer review or human experimentation committee to oversee his work. It was his decision to experiment with this therapy. This also meant that distinguishing between what was an experimental therapy and what was a "true" therapy was virtually indistinguishable with regard to multiple sclerosis in the 1930s, and as will be seen in chapter five, through the 1950s. That this was unique to the NYNI seems doubtful. Frederick Tilney noted with satisfaction in 1933 that "the Brickner Quinine Treatment for Multiple Sclerosis is now being widely used throughout the country . . . and many others are using it as a routine measure of therapy."[91] The clinic became a site of experimental physiology where the individual physician's knowledge of biochemistry, physiology, anatomy, and pathology served as the scientific basis of clinical practice. In this structure of decision making it was the individual physician who decided efficacy not abstract statistics or a peer review committee. The key factor that created a blurry boundary between the laboratory and the clinic and between experiment and therapy was that the individual researcher held the power for managing these boundaries. The Commonwealth Fund's granting mechanisms supported this practice at the NYNI.

As a specialty neurology had limited ability to control knowledge production about multiple sclerosis given the decentralized structures and sources of funding for neurological research during the period before the late 1940s and early 1950s. In 1946 a new impulse toward reorganizing and nationalizing the structure of multiple sclerosis research emerged in New York City. The antagonism which developed between the National Multiple Sclerosis Society and the Rockefeller Foundation and the Commonwealth Fund underscores how the structure of funding medical research shaped the culture of research conducted before 1946.

In 1947 the Association for the Advancement of Research in Multiple Sclerosis (AARMS, changed to the National Multiple Sclerosis Society in 1948) asked the

Commonwealth Fund for twenty thousand dollars. The AARMS wanted money for their own organization and for the Neurological Institute to do work on multiple sclerosis. The AARMS did not know that the Commonwealth Fund had given over $100,000 for research on multiple sclerosis at the Neurological Institute from 1920 to 1934 and from 1942 to 1945.[92] Barry C. Smith, General Director of the Commonwealth Fund, rejected the request saying that the "Commonwealth Fund prefers to deal directly with the institution of people conducting research financed by it and I am sorry to have to tell you that the Fund is not interested in adding anything to the appropriation for supervisory or other expenses of the" AARMS.[93] Dr. Willard C. Rappleye, Dean, College of Physicians and Surgeons, Columbia University, concurred saying that "we do not need an intermediary broker."[94] In 1949 the NMSS asked the Rockefeller Foundation for a grant which was also rejected. The NMSS wanted to be a national clearing-house through which all MS funding would be channelled so that research could be coordinated to over come the disconnected vectors of research structured by local systems of foundation and university funding. As Vermont professor George A. Schumacher commented in 1952: "impetus for the first great wave of concentrated effort and research on a wide scale came from a group of laymen who in 1946 founded the National Multiple Sclerosis Society . . . When the available facts and hypotheses were assembled, it became evident that there was n meeting of minds on the questions of etiology, pathogenesis, or treatment in multiple sclerosis."[95] In the late 1940s the National Multiple Sclerosis Society replaced the earlier foundations as the primary source of support for research on MS and in 1949 began lobbying the federal government for additional research outlays on multiple sclerosis. The point of this last story is to underscore the culture of research in the older model of funding multiple sclerosis research. A new model emerged from 1946 to 1952 which will be taken up in chapter four where I will analyze the creation and

activities of the National Multiple Sclerosis Society.

V Conclusion

In conclusion the boundaries between social and neurological questions remained blurry through the 1920s in the United States because of underdeveloped state of the specialty of neurology and because of the limited number of tools available for would be neurological researchers. By the 1920s, neurologists had created new institutions in the United States such as the New York Neurological Institute and the Association for Research in Nervous and Mental Diseases. These new specialty structures became equipped with new tools such as the lumbar puncture and biochemistry which allowed neurological investigations to go in new directions. Even though the new laboratory work seemed increasingly distant from social questions, the motive of the Commonwealth Fund was quick results which might result in economic benefit to the nation. The motives of the neurologists, while they overlapped with the Commonwealth Fund, diverged as well. Specifically neurologists were interested in their own interests and careers, building up their own institution, and in building better scientific foundations for their specialty in the unique context of the United States. Neurologists were able to carry out research on multiple sclerosis in the 1920s through the 1940s because of an expansion of funding by private foundations in the United States which changed the economy of practice by allowing neurologists some freedom from purely clinical work which had been the financial foundation of their specialty. Nevertheless, the way in which the Commonwealth Fund dispersed funds to the New York Neurological Institute researchers encouraged highly autonomous work by individual researchers working on the same disease. This led to medical research where the boundaries between the clinic and the laboratory and between experimentation and therapy were barely distinguishable. It also led to a national pattern of research on multiple sclerosis in the 1920s through the

ENDNOTES

1. Pnina G. Abir-Am, "'New'" trends in the history of molecular biology," *Historical Studies in the Physical Sciences* 26 (1995): 177-89. See Robert Kohler, *Partners in Science: Foundations and Natural Scientists* (Chicago: University of Chicago Press, 1991). Lily E. Kay, *The Molecular Vision of Life: Caltech, the Rockefeller Foundation, and the Rise of the New Biology* (New York: Oxford University Press, 1993). E. Richard Brown, *Rockefeller Medicine Men: Medicine and Capitalism in America* (Berkeley: University of California Press, 1979).

2. J.K. Bauduy, "Clinical Lecture Multiple Cerebro-Spinal Sclerosis," *Missouri Clinic Recorder* 1 (1874): 3-5.

3. C.H. Boardman, "Progressive Multiple cerebro-spinal sclerosis," *The Northwestern Medical and Surgical Journal* 3 (1873): 252.

4. Jonathan Hutchinson, *The Pedigree of Disease; Being Six Lectures on Temperament, Idiosyncrasy, and Diathesis* (London, 1884), 71, quoted in Charles E. Rosenberg, "The Bitter Fruit: Heredity, Disease, and Social Thought," in *No Other Gods: On Science and American Social Thought* (Baltimore: The Johns Hopkins University Press, 1976), 29.

5. Erwin H. Ackerknecht, "Diathesis: The Word and the Concept in Medical History," *Bulletin of the History of Medicine* 56 (1982): 317-325.

6. Ibid.

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Modernity: Max Nordau as a Critic of *Fin-de-Siècle* Modernism," *German Studies Review* 14 (1991): 475-82. Dain Borges, "'Puffy, Ugly, Slothful and Inert': Degeneration in Brazilian Social Thought, 1880-1940," *Journal of Latin American Studies* 25 (1993): 236-39. Roberty A. Nye, "Degeneration, Hygiene and Sports in *Fin-de-Siècle* France," *Proceedings of the Annual Meeting of the Western Society for French History 1980* 8 (1981): 406-7. Rosenberg, "The Bitter Fruit: Heredity, Disease, and Social Thought," 25-53.

13. George T. Stevens, *Functional Nervous Diseases: Their Causes and Their Treatment* (New York: Appleton and Company, 1887), 15-16. Stevens wrote the essay as a student at the Academie Royale de Medicine de Belgique. It is probable that he encountered the French degenerationist theories there.

14. Ibid., 6.

15. James J. Putnam, "A Group of Cases of System Scleroses of the Spinal Cord . . .," 70.

16. Charles L. Dana, *Textbook of Nervous Disease* (New York: William Wood and Company, 1892), 374.

17. Ibid., 374. At the time Dana was Professor of Nervous and Mental Diseases at the New York Post-Graduate Medical School; Professor at Dartmouth Medical College; Visiting Physician and Bellevue Hospital; Neurologist to the Montefiore Home; and ex-president of the American Neurological Association.

18. 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. Neff and Klingmann, "A Case of Multiple Cerebro-Spinal Sclerosis of a Special Anatomical Form," 431-433.

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20. 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): 431-42.

21. Ellen Dwyer, "Stigma and Epilepsy," *Transactions & Studies of the College of Physicians of Philadelphia* 13 (1991): 397, 401.

22. Dr. Joseph Fraenkel presented a case of congenital multiple sclerosis at the New York Neurological Society in 1902, Society Proceedings, *JNMD* 30 (1903): 215. 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."Smyth Ely Jelliffe, "Multiple Sclerosis its Occurrence and Etiology," *Journal of Nervous and Mental Diseases* 31 (1904): 453. Dr. I. Abrahamson again before the New York Neurological Society in 1915 presented two cases of multiple sclerosis in a mother and son. I. Abrahamson, "A Multiple Sclerosis in Mother and Son," *JNMD* (1915): 295. As late as 1935, Philadelphia physician Alfred Gordon writing in the *Eugenical News* in 1935 argued that with respect to multiple sclerosis, "we are therefore dealing here with some congenital defects of the nervous system transmitted to some descendant in the form of Disseminated Sclerosis." Alfred Gordon, "The Problems of Heredity and Eugenics," *Eugenical News* 20 (July-August 1935): 52.

23. William A. White, " Eugenics and Heredity in Nervous and Mental Diseases," in *The Modern Treatment of Nervous and Mental Diseases,* ed. William A. White and Smith Ely Jelliffe (Philadelphia and New York: Lea & Febiger, 1913), 17-19.

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25. Ibid., 38.

26. T.H. Weisenburg, "Multiple Sclerosis; its occurrence in families: brother and sister," *Archives of Diagnosis* 2 (1909): 167-74.

27. "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.

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

29. Rosenberg, "Bitter Fruit," 44-53; Bonnie Ellen Blustein, "New York Neurologists and the Specialization of American Medicine," *Bulletin of the History of Medicine* 53 (1979):176-183. Michael F. Guyer, Professor of Zoology, University of Wisconsin, wrote 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 far-reaching effects," Michael F. Guyer, *Being Well-Born: an Introduction to Heredity and Eugenics* (New York: Bobbs-Merrill Company, 1927, c1916), 339; Samuel J. Holmes, Professor of Zoology, University of California, Berkeley, 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," 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.

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

31. Thomas W. Salmon, "Immigration and the Mixture of Races in Relation to the Mental Health of the Nation," in *The Modern Treatment of Nervous and Mental Diseases,* 243.

32. Ibid., 245.

33. "The Irish were of different but not greatly dissimilar racial origin and they had lived for centuries in close relations with one of the colonial racial stocks, speaking the same language and frequently intermarrying," ibid., 248-251, 257, 259.

34. Ibid., 252, 259.

35. Ibid., 255.

36. Ibid., 258.

37. Ibid., 258, 274.

38. Ibid., 269.

39. W.N.P. Barbellion, *The Journal of a Disappointed Man,* with an introduction by H.G. Wells (London: Chatto & Windus, 1919), 226. This example is from a young Englishman from London. Even though it is not an American example I think the evidence for the United States suggests that this would have been a typical reaction considering the wide currency of eugenic ideas in the American middle classes. Unfortunately I do not have an American diary for this period. For the history of the eugenics movements in the United States and Great Britain and its relatively greater

influence in the United States see Daniel J. Kevles, *In The Name of Eugenics: Genetics and the Uses of Human Heredity* (Berkeley: University of California Press, 1985).

40. Typescript interview of Dr. Bernard Glueck by Barbara S. Quinn, Asst. Director, The Commonwealth Fund, NYC, 4/18/1927, Rockefeller Archive Center, Collection Commonwealth Fund <hereafter> RAC/CF, Series 1, Box 234, Folder 2220.

41. On February 10, 1920 New York Neurological Society members Drs. Walter Timme, Charles Dana, Frederick Tilney, J. Ramsay Hunt, Smith Ely Jelliffe, Foster Kennedy, Bernard Sachs founded the Association for Research in Nervous and Mental Diseases. The group was mainly for neurologists and neurological questions. See "A Brief History of the ARNMD," Box 27, Folder "A Brief History of the ARNMD," *American Neurological Association Archives,* Bowman-Gray Medical School, Winston-Salem, North Carolina.

42. Discussion of Smith Ely Jelliffe, "The Parts of the Central Nervous System Which Tend to Exhibit Morbid Recessive or Dominant Characters,"*Heredity in Nervous and Mental Disease: An Investigation by the Association for Research in Nervous and Mental Disease* (New York: Paul B. Hoeber, 1925), 62-76, 99-100.

43. ARNMD Commission, "The Conclusions of the Commission," *Multiple Sclerosis* (1922), 47. ARNMD Commission, "Conclusions of the Commission Concerning the Pathology of Multiple Sclerosis," *Multiple Sclerosis* (1922), 208.

44. "Comments of Dr. Casamajor to AG re NY Neurological Institute, May 1936," from Allan Greg's Diary, Director, The Medical Sciences,The Rockefeller Foundation, RAC, Collection Rockefeller Foundation <hereafter RAC/RF>, Series 200, Box 79, Folder 951. Dr. Heffron, CF, "Columbia-Presbyterian Medical Center-Neurological Institute- Research in Neurological Diseases- Review of project and summary memorandum," 3/14/1945," 2, RAC/CF, Series 18.1, Box 79, Folder 724.

45. Dr. Heffron, Commonwealth Fund <hereafter CF>, "Columbia-Presbyterian Medical Center-Neurological Institute- Research in Neurological Diseases- Review of project and summary memorandum," 3/14/1945," 3, RAC/CF, Series 18.1, Box 79, Folder 724.

46."CF Grants Series 1, Neurological Institute, Multiple Sclerosis, Epilepsy, and Social Research," 9/30/1919 to 12/29/1922, RAC/CF, Box 234, Folder 2218. Drs. Joseph Collins, Joseph Frankel, and Pearce Bailey founded the NYNI in 1909. In 1925 the NYNI became affiliated with Columbia University College of Physicians and Surgeons. The Presbyterian Hospital, the Babies Hospital, and the Neurological Institute merged into one corporation in 1937. "Neurological Institute Collection Description," Archives and Special Collections, Health Science Library, Columbia University, New York, NY.

47. "Neurology," 5/7/1920, *RAC/CF*, Series 1, Box 234, Folder 2218." "Neurology, Presented to Directors 6/26/20," p. 5, RAC/CF, Series 1, Box 234, Folder 2218

48. "No. 73 The Neurological Institute," 12/3/1919, RAC/CF, Series 1, Box 234, Folder 2218.

49. Ibid. In 1919 the most prominent physicians associated with the NYNI were Charles A. Elsberg, Charles Dana, Pearce Bailey, Ramsey Hunt, and Frederick Tilney.

50. "Neurological Institute. Interview with Dr. Longcope Wednesday, May 26, 1920," RAC/CF, Series 1, Box 234, Folder 2218.

51. Ibid. "Neurology, Presented to Directors 6/26/20," RAC/CF, Series 1, Box 234, Folder 2218.

52. "Neurology, Presented to Directors 6/26/20," 5-6, RAC/CF, Series 1, Box 234, Folder 2218.

53. Letter, Robert Thorne, Esq. NYC to Professor Max Farrand, CF, NYC,

10/11/1920, 1, in RAC/CF, CF Grants Series 1, Neurological Institute, Box 234, Folder 2218. Letter, Drs. Charles A. Elsberg and Frederick Tilney, NYC to Max Farrand, CF, NYC, 10/18/1920, RAC/CF, Series 1, Box 234, Folder 2218.

54. Letter, <not signed but presumed to be Max Farrand> CF, NYC to Drs. Charles A. Elsberg and Frederick Tilney, NYC, 10/28/1920, RAC/CF, Series 1, Box 234, Folder 2218.

55. Charles E. Rosenberg, "Introduction: Framing Disease: Illness, Society, and History," in *Framing Disease: Studies in Cultural History*, ed. Charles E. Rosenberg and Janet Golden (New Brunswick, NJ: Rutgers University Press, 1992), p. xxi.

56. L.H. Cornwall, "Report of Multiple Sclerosis Research for the period October 1, 1923 to September 30, 1924," 4, 9/30/1924, RAC/CF, Series 1, Box 234, Folder 2219.

57. Letter, Frederick Tilney, NYC, to Max Farrand, CF, NYC 1/18/1921, RAC/CF, Series 1, Box 234, Folder 2218. Letter, Frederick Tilney, NYC, to Max Farrand, CF, NYC 1/19/1921, RAC/CF, Series 1, Box 234, Folder 2218.

58. Doctor Frederick Tilney, Dept. of Neurology, Columbia University, "Report on the Research in Multiple Sclerosis," 4/17/1929, RAC/CF, Series 1, Box 234, Folder 2220. Letter, Doctor Frederick Tilney, NYC, to Barbara S. Quinn, Asst. Director, CF, NYC to Dr. Theodore Weisenburg, Philadelphia, PA, 4/6/1928, RAC/CF, Series 1, Box 234, Folder 2220.

59. Letter, Barbara S. Quinn, Asst. Director, CF, NYC to Dr. M.C. Winternitz, Sterling Hall of Medicine, Yale University, 3/29/1927, RAC/CF, Series 1, Box 234, Folder 2220.

60. Letter, Dr. M.C. Winternitz, Sterling Hall of Medicine, Yale University to Barbara S. Quinn, Asst. Director, CF, NYC, 3/25/1927, RAC/CF, Series 1, Box 234, Folder 2220.

61. Ibid.

62. Letter, Dr. M.C. Winternitz, Sterling Hall of Medicine, Yale University to Barbara S. Quinn, Asst. Director, CF, NYC, 3/30/1927, RAC/CF, Series 1, Box 234, Folder 2220.

63. Letter, Barbara S. Quinn, Asst. Director, CF, NYC to Dr. M.C. Winternitz, Sterling Hall of Medicine, Yale University, 3/29/1927, RAC/CF, Series 1, Box 234, Folder 2220.

64. Letter, Barbara S. Quinn, Asst. Director, CF, NYC to Dr. Theodore Weisenburg, Philadelphia, PA, 4/4/1927, RAC/CF, Series 1, Box 234, Folder 2220.

65. Letter, Albert M. Barrett, M.D., Director, State Psychopathic Hospital, University of Michigan, Ann Arbor, MI to Barbara S. Quinn, Asst. Director, The Commonwealth Fund, NYC, 4/18/1927, RAC/CF, Series 1, Box 234, Folder 2220. For a similar formulation see also typescript interview of Dr. T.H. Ames by Barbara S. Quinn, Asst. Director, The Commonwealth Fund, NYC, 4/20/1927, RAC/CF, Series 1, Box 234, Folder 2220.

66. Letter, Dr. Hugh T. Patrick, Chicago, IL to Barbara S. Quinn, Asst. Director, The Commonwealth Fund, NYC, 4/6/1927, RAC/CF, Series 1, Box 234, Folder 2220.

67. "#1918. Neurological Institute- Studyof Epilepsy and Multiple Sclerosis," Presented to the Directors of the CF at a meeting on 6/8/1927, RAC/CF, Series 1, Box 234, Folder 2220.

68. "#2051. Neurological Institute- Study of Epilepsy and Multiple Sclerosis," Presented to the Directors of the CF at a meeting on 6/5/1928, RAC/CF, Series 1, Box 234, Folder 2220.

69. L.H. Cornwall, "Multiple Sclerosis Investigation," 9/15/1928, RAC/CF, Box 234, Folder 2220. Doctor Frederick Tilney, Dept. of Neurology, Columbia University,

"Report on the Research in Multiple Sclerosis," 4/17/1929, RAC/CF, Series 1, Box 234, Folder 2220.

70. " 2821 Neurological Institute-Study of Multiple Sclerosis Interview with Doctor Frederick Tilney and Dr. Richard Brickner," NYC, By E.K. Wickman, 5/8/1934, RAC/CF, Series 1, Box 234, Folder 2223.

71. Letter, Doctor Frederick Tilney, NYC, to E.K. Wickman, CF, NYC 4/27/1931, RAC/CF, Series 1, Box 234, Folder 2221.

72. Letter, Doctor Frederick Tilney, NYC, to E.K. Wickman, CF, NYC 5/9/1932, RAC/CF, Series 1, Box 234, Folder 2222.

73. Memorandum, E.K. Wickman, CF to Mr. Smith, CF, 5/14/1934, RAC/CF, Series 1, Box 234, Folder 2223.

74. "Neurological Institute, Study of Multiple Sclerosis, Interview with Dr. Leon H. Cornwall," 5/19/1934, RAC/CF, Series 1, Box 234, Folder 2223.

75. Ibid.

76. Letter, Barry C. Smith, CF, NYC to P.W. Bunnell, NYC, 6/9/1939, RAC/CF, Series 1, Box 234, Folder 2223.

77. From Allan Greg's Diary, 11/25/1938, Director, The Medical Sciences, RAC/RF, Record Group 1.1, Series 200, Box 79, Folder 951. Memorandum of Allan Greg's, interview with Tracy Putnam, 11/30/1938, RAC/RF, Record Group 1.1, Series 200, Box 79, Folder 951.

78. See Tracy Jackson Putnam, "Curriculum Vitae," and "Biographical Sketch," *Tracy Jackson Putnam Collection,* Louise Darling Biomedical Library, University of California, Los Angeles, Box 1, Folder Biographical Materials.

79. Tracy Jackson Putnam, M.D., "Publications," ibid.

80. Letter, Tracy J. Putnam, NYC, to Dean Sage, Presbyterian Hospital, NYC, 11/21/1941, RAC/CF, Series 18.1, Box 79, Folder 723.

81."Columbia University, College of P. & S., Neurological Institute. Research in Neurological Diseases. Interview with Dr. Tracy Putnam, by Dr. Roderick Heffron," 1/2/1942, RAC/CF, Series 18.1, Box 79, Folder 723.

82. Letter, Tracy J. Putnam, NYC, to Dean Sage, Presbyterian Hospital, NYC, 11/21/1941, RAC/CF, Series 18.1, Box 79, Folder 723.

83. Memorandum, Dr. R. Heffron to Mr. Barry C. Smith, CF, NYC, 3/29/43, RAC/CF, Series 18.1, Box 79, Folder 723.

84. Dr. Heffron, "Review of Project with Dr. Tracy Putnam and Summary Memorandum, 3/20/1944,"16, RAC/CF, Series 18.1, Box 79, Folder 723.

85. "Excerpt from RAL diary of November 9, 1945," RAC, Collection RF, Record Group 1.1, Series 200, Box 79, Folder 952.

86. "Excerpt from RS Morison diary of November 21, 1945," Dr. Willard C. Rappleye, Dean, College of Physicians and Surgeons, Columbia University, RAC/RF, Record Group 1.1, Series 200, Box 79, Folder 952.

87. Letter, Tracy J. Putnam, NYC to Dean Willard C. Rappeley, College of Physicians and Surgeons, 6/30/1947, RAC/CF, Series 18.1, Box 79, Folder 724.

88. Dr. Heffron, "Review of Project with Dr. Tracy Putnam and Summary Memorandum, 3/20/1944," RAC/CF, Series 18.1, Box 79, Folder 723.

89. Leon H. Cornwall, "Report concerning the Investigation of Multiple Sclerosis in Columbia University and the New York Neurological Institute Under a Grant from the Commonwealth Fund," late 1926 or early 1927, RAC/CF, Series 1, Box 234, Folder 2219.

90. Patient 3, Neurological Exam, 4/3/19, *New York Hospital/Cornell Hospital Archives*. Patient 9, 11/20/1925, ibid. Patient 5, 7/28/1926, ibid. Patient 6, 5/281926, ibid.

91. Letter, Doctor Frederick Tilney, NYC, to CF, NYC, "Report on Research in Multiple Sclerosis," 3-4, 5/13/1933, RAC/CF, Series 1, Box 234, Folder 2222.

92. Letter, Carl M. Owen, President, Association for Advancement of Research on Multiple Sclerosis, NYC, to Barry C. Smith, General Director, CF, 4/8/1947, RAC, Series 18.1, Box 79, Folder 724.

93. Letter, Barry C. Smith, General Director, CF, to Carl M. Owen, President, Association for Advancement of Research on Multiple Sclerosis, NYC, 4/10/1947, RAC/CF, Series 18.1, Box 79, Folder 724.

94. Letter, Dr. Willard C. Rappleye, Dean, College of Physicians and Surgeons, Columbia University, NYC, to Barry C. Smith, CF, 4/22/1947, RAC/CF, Series 18.1, Box 79, Folder 724.

95. George A. Schumacher, "Forward: Symposium on Multiple Sclerosis and Demyelinating Diseases," *The American Journal of Medicine* 12 (1952): 499-500.

Chapter Four
The Emergence of Ms as a Research Priority and a Popular Crusade, 1946-1960

I Introduction

From the 1870s to the 1910s, American neurologists considered MS a rare malady in the United States. During the 1920s they began to diagnose more MS cases; so that by the early 1930s American neurologists considered MS a common neurological disease in the United States. Nevertheless MS was not an object of intense scientific scrutiny in the 1930s and early 1940s though some prominent American medical scientists were studying the disease. For the lay public, MS remained a virtually unknown disease during this same time period. Suddenly after 1946 scientific research on MS and the public's interest in it increased dramatically.

Table 1 shows the production of knowledge about MS in the United States as measured in the total number of articles written on MS in American medical journals in five year periods from 1870 to 1959.[1] Neurologists demonstrated increased interest in MS during the 1930s and early 1940s at least as expressed in the numbers of articles published on the subject in American medical journals. Beginning in 1947 there was a sharp rise in the number of articles devoted to MS. This reflected an intensification of experimental research and clinical interest in the disease in the United States.

This new concern with MS resulted in several scientific conferences. On December 10 and 11, 1948 the Association for Research in Nervous and Mental Diseases (ARNMD) devoted their annual conference exclusively to MS. The New York Academy of Sciences held a conference on April 17 and 18, 1953 entitled, "The Status of Multiple Sclerosis," which was chaired by Pearce Bailey, Director of the National Institute for Neurological Diseases and Blindness (NINDB).[2] In 1957 the National Multiple Sclerosis Society (NMSS) co-sponsored a symposium entitled, "New Research Techniques of

Table 1

Number of Articles in Five Year Periods on MS in Biomedical Literature of USA

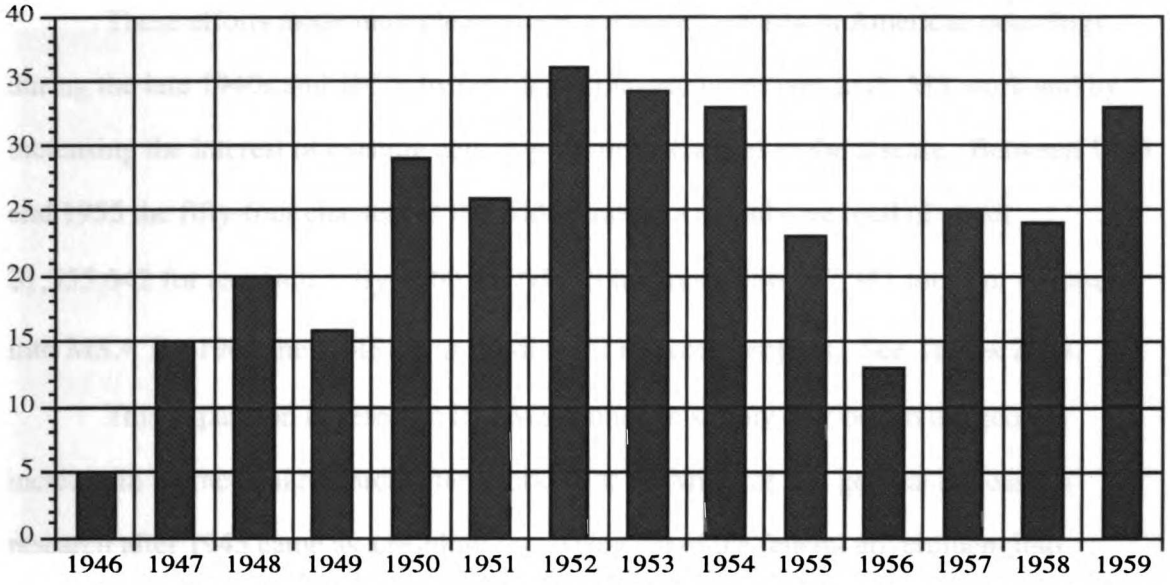
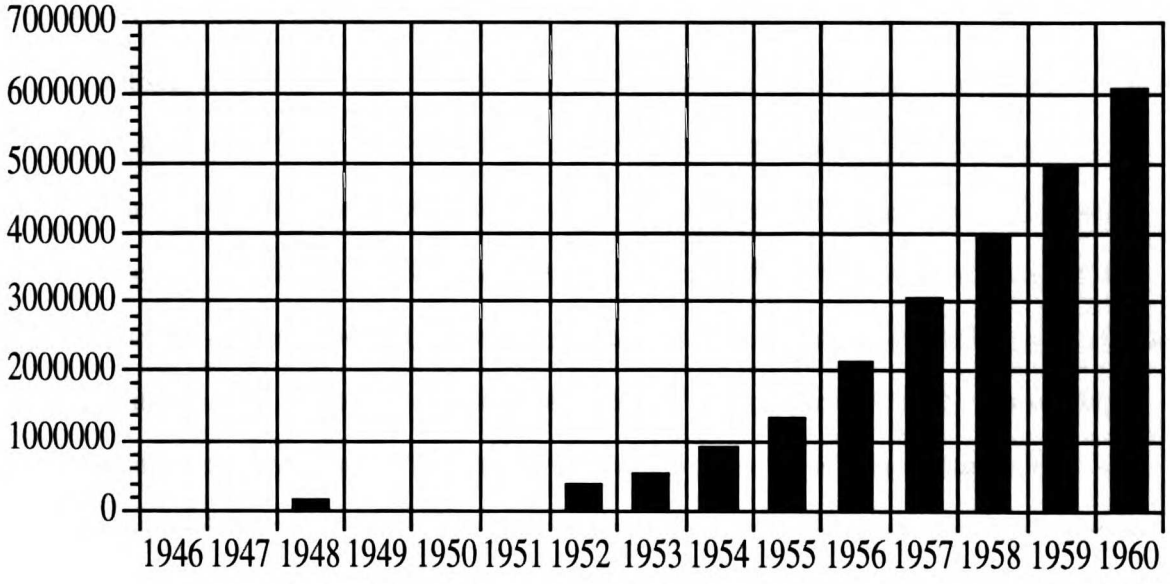


Table 2

NMSS Annual Cumulative Research Dollars
(Figures not available for 1946-1947, 1949-1951)



Neuroanatomy."[3] In 1962, the NMSS co-sponsored a conference at the University of California, Los Angeles on the "Mechanisms of Demyelination."[4]

These efforts made multiple sclerosis a research priority in American neurology during the late 1940s and 1950s by creating economic incentives to do MS work and by increasing the interest of existing neurological organizations in the disease. Between 1946 and 1955 the fifty-four chapters of the NMSS raised a cumulative total of about $1,355,642 for research.[5] By 1960 the NMSS had raised $6,067,381 total for research into MS.[6] By 1960 the NMSS was funding 72 research projects. See Tables 2,3,4.

This expansion of research into MS cannot be simply ascribed to the general increase in biomedical research after World War II. Much of this general increase in research after 1945 came as a result of the expansion of the federal government into biomedical research. However, the federal government did not fund neurological research in any signficant way until 1952. The expansion of research into MS predated the federal interest in the disease. An intensification of research in MS began in 1947 and was in full stride by 1951. See Table 5.

Observers at the time noted the increased interest in the disease. Vermont professor George Schumacher noted in 1952 that the "general awareness among medical scientists and practioners of the great prevalence of multiple sclerosis has been a surprisingly recent development in the history of the study of this disease. Although isolated investigators in the past quarter century devoted much energy and time toward an understanding of multiple sclerosis, it remained an esoteric problem of medicine generally neglected by the medical scientist and clinician alike . . . Impetus for the first great wave of concentrated effort and research on a wide scale came from a group of laymen who in 1946 founded the National Multiple Sclerosis Society . . ."[7] Other medical observers noted the sharp increase in interest about MS.[8] In 1952 physician Cornelius Traeger noted the "renaissance of interest

Table 3

Annual Research Budget NMSS
(Figures not available for 1946-1949, 1951)

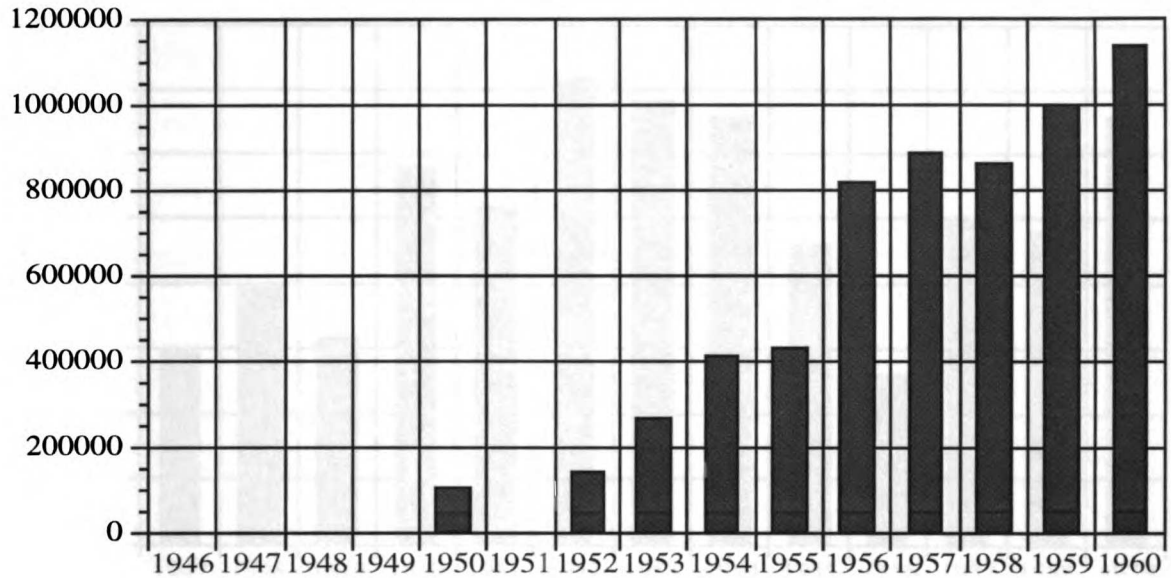


Table 4

Number of Research Projects Supported by the NMSS Per Year

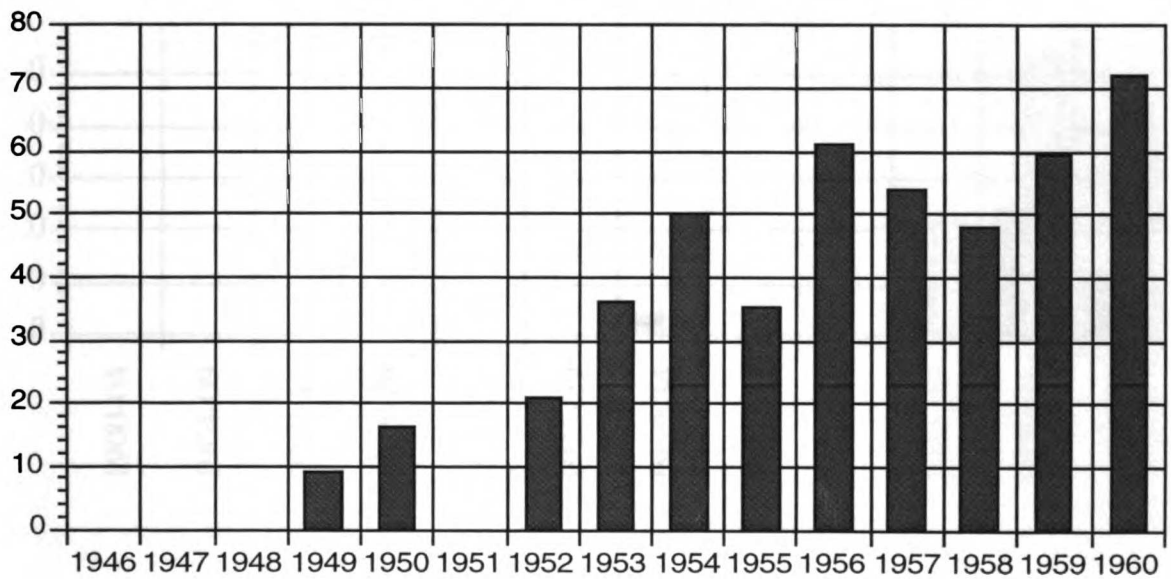


Table 5

Number of Articles Total on MS from 1946 to 1959 Per Year in Biomedical Literature of USA

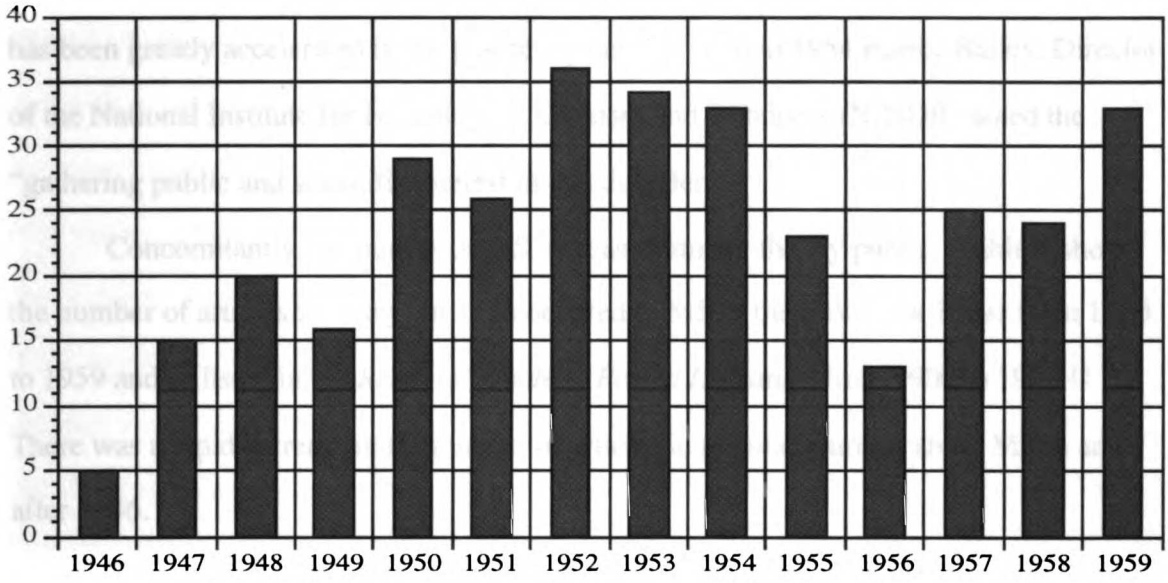
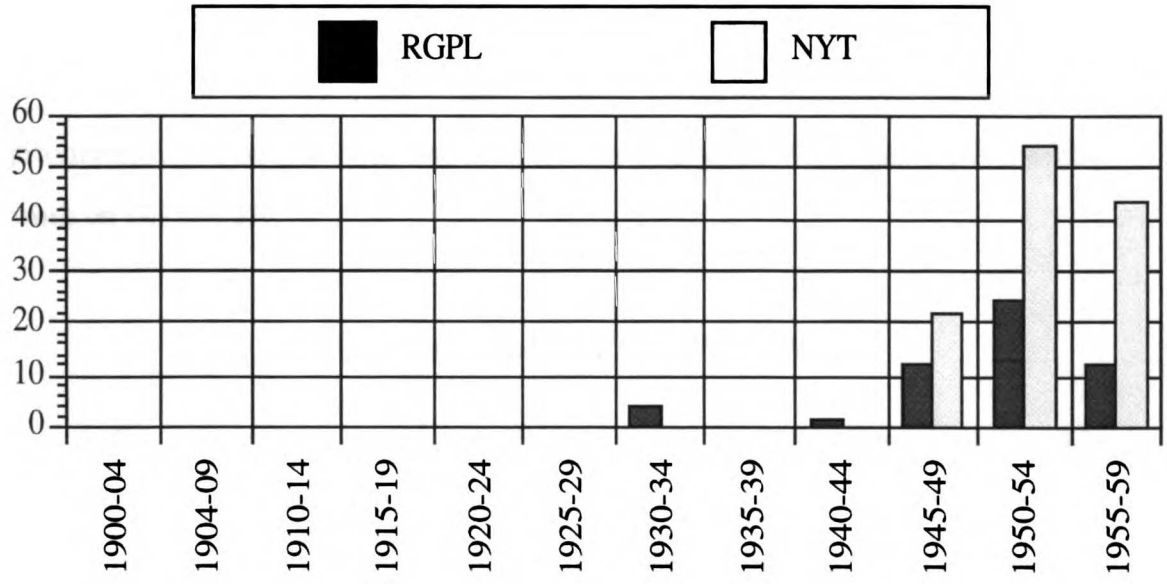


Table 6

Number of Articles Total in Five Year Periods On MS in Popular Literature of USA



in the problem of multiple sclerosis and the demyelinating diseases and the widespread increased activity in this field of research."[9] In 1954 W.H. Sebrell, Jr., Director of the National Institutes of Health (NIH), wrote that "the research attack on multiple sclerosis has been greatly accelerated in the past few years."[10] Also in 1954 Pearce Bailey, Director of the National Institute for Neurological Diseases and Blindness (NINDB) noted the "gathering public and scientific interest in this disorder."[11]

Concomitantly, awareness of MS increased among the lay public. Table 6 shows the number of articles in five year totals devoted to MS in the *New York Times* from 1930 to 1959 and as listed in the *Readers' Guide to Period Literature* from 1900 to 1959.[12] There was a rapid increase in the number of articles in popular journals about MS in and after 1946.

What explains this sudden increase in medical activity concerning MS and the growing awareness of the disease by the lay public especially considering there was no dramatic epidemic of MS and no evidence that it was an infectious condition?[13] The answer is that MS patients, their families, their partisans put MS on the cognitive map of American medicine and popular culture. In other words, lay activists made MS a research priority in American neurology and were directly responsible for the increase in scientific work on this malady.

II The National Multiple Sclerosis Society

The story of lay activism and MS began in 1945 with two New Yorkers in their twenties. Sylvia Lawry was becoming increasingly distraught because of the decline of her brother Bernard Friedman, a victim of MS. Bernard's doctor would refuse to see him when a new symptom occurred unless it was catastrophic. The physician's advice, not unusual for the time, was to go home and rest because there was nothing medicine could do

for this disease. Because of this attitude Lawry began reading the medical literature for herself hoping to find some clue about treatment in order to help her brother. She discovered that many MS patients experienced remissions; so she decided to investigate the factors which might precipitate a remission. To do this she placed a personal advertisement in the *New York Times* in May 1945 which read: "Multiple Sclerosis. Will anyone recovered from it please communicate with patient. T272 Times." Lawry received about fifty replies, mostly from patients. She and the respondents continued to correspond about the problems surrounding multiple sclerosis. They then began to hold meetings at the New York Academy of Medicine and at the Red Cross headquarters in New York City. They decided to start a national organization dedicated to finding a cure for MS. The New York Academy of Medicine then donated office space to the fledgling group. In March 1946, patients, their families, and their partisans formed the Association for the Advancement of Research into Multiple Sclerosis (AARMS).[14] In 1948 the group reorganized and named itself the National Multiple Sclerosis Society (NMSS). In 1946 the AARMS gave their members, mostly patients, stacks of cards to enroll the patients' families and friends in the new organization; because of this Lawry described the patients as the "prime movers" in the new organization.[15] They placed an advertisement in a Boston paper that attracted more potential members.[16] Table 7 shows that membership quickly rose after 1946. In 1946 the AARMS had 600 members, a figure which grew to 7,500 in 1948. In 1949 the NMSS membership had climbed to 15,000. By 1954 the organization had 33,000 members and in 1958 the figure rose to 120,000.[17]

For the AARMS, Lawry recruited a lay Board of Directors and a Medical Advisory Board. For the lay board she wanted professionals and business people whose prominence and influence was national in scope. Raymond Moley, contributing editor at *Newsweek*, became the first Chairman of the Public Education Committee and chaired the first press

Table 7

Number of Members of NMSS Per Year
(Figures not available for 1947, 1950-1952, 1955-1957, 1959-1960)

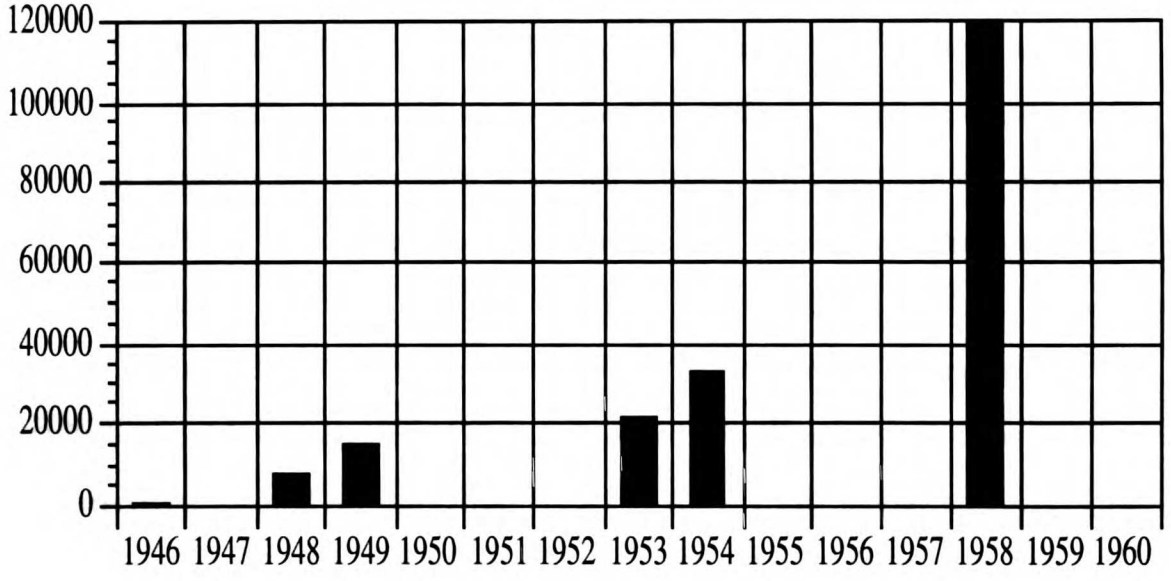
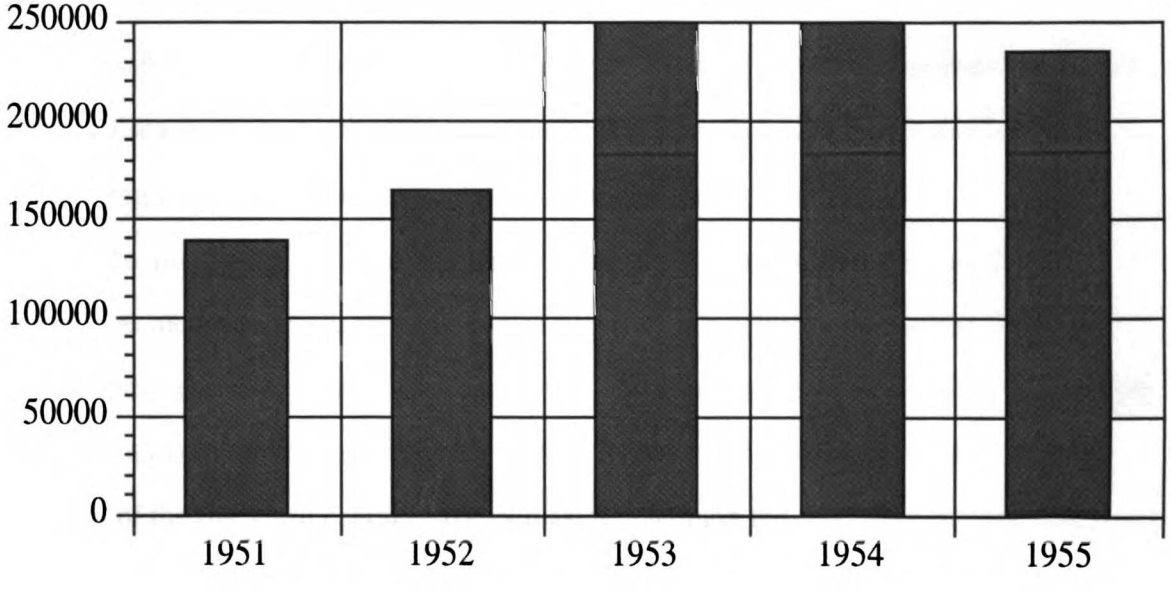


Table 8

Annual Dollars Spent by the NINDB on MS
(The 1955 figure represents through August 1955 only.)



conference of the AARMS. Carl W. Owen of the law firm of Owen, Willkie, Otis, Farr and Gallagher became Chairman of the Board of Directors.[18] Other prominent sponsors included: Mrs. James S. Rockefeller; Mrs. Wendell Wilkie; William J. Norton, Secretary of the Children's Fund of Michigan; and Senator Brien McMahon of Connecticut.[19]

To select a Chair for the Medical Advisory Board, Lawry read the medical literature to find the most prominent researcher in multiple sclerosis in the United States. Richard Brickner and Tracy Jackson Putnam were two of the most prominent researchers in MS in the USA during the 1930s and 1940s. Lawry chose Putnam to be the first chair of the Medical Advisory Board because he was director of the New York Neurological Institute and Professor of Neurosurgery and Neurology at Columbia University.[20] Other prominent members of the Medical Advisory Board included: Roger I. Lee, retiring president of the AMA; Thomas M. Rivers, Director of the Hospital of the Rockefeller Institute for Medical Research; Dr. Ernest L. Stebbins, Dean of the School of Hygiene of Johns Hopkins University; Henry Woltman of the Mayo Clinic; and Leo Alexander of Boston.[21] Lawry had rapidly put together lay and medical boards with very prominent members. What explains the suddenness of the emergence of the MS movement in 1946? Why was Lawry so successful so quickly considering the apparent obscurity of MS as compared with well-known diseases such as polio and cancer, diseases for which activists staged massive advertising campaigns from the mid-1940s through the 1950s.[22]

To answer this question it is important to remember from chapter two that physicians increasingly diagnosed patients with multiple sclerosis during the 1930s and 1940s compared with decades previous to this. In effect, this created a significantly larger patient population with this incurable chronic disease than had existed in the first three decades of the twentieth century. By increasingly naming the disease physicians, but especially neurologists, created the potential for a patient social movement centered on MS.

Lawry found fertile ground on which to build a new organization because of the youth of many of the MS patients, because little could be done for them, because medicine seemed to be ignoring their plight, and because of the patients' desperateness. Also, because the life expectancy of MS patients was only slightly less than average, the increasing tendency of neurologists to diagnose multiple sclerotics from the early 1930s onwards meant there was a snowball effect in terms of patient numbers which reached a critical mass by 1946. In short, there were simply more MS patients. Lawry's efforts helped overcome the isolation of many MS sufferers and tapped into a deeply felt need by patients for an organization to address what they saw as a lacuna in modern medical research and treatment. Also, by 1946 many prominent figures in American society knew someone with MS. Raymond Moley, editor of *Newsweek*, had two promising students come down with MS while he was teaching at Barnard College. Senator Charles Tobey of New Hampshire had a daughter with the disease. Henry Kaiser, Jr., son of the industrialist, was also a MS sufferer.[23]

This newly created pool of MS patients in the 1940s coincided with a political and cultural climate highly favorable to disease crusades and the private and public outlay of funds for biomedical research. Moreover, American neurologists, a relatively low status and poorly funded specialty in the United States in the mid-1940s, quickly realized the benefits a vigorous patient movement could mean for their specialty.

These conditions made possible the successful efforts of MS patients, their families, their partisans, and their neurological allies through the NMSS to make MS a research priority for American neurology in the 1950s. They did this by raising large sums of money for research directly and indirectly by persuading the federal government to spend money on MS research. The NMSS carried the campaign for dollars, for public awareness, and for the attention of the medical profession on two main fronts. On the first front the NMSS carried on a media campaign that attempted to raise public consciousness

about MS and that helped raise money. On the second front the NMSS vigorously lobbied the federal government to fund and carry out research on MS.

III The First Front: Advertising Disease

Part of the problem the MS activists faced in the late 1940s was public ignorance about the disease unlike the situation with cancer or polio.[24] As an AARMS pamphlet stated in 1946: "to the man in the street, the term multiple sclerosis is strange and unfamiliar. The time has come when the world should know about multiple sclerosis for this widely prevalent nerve disease has become an acute social problem. Only the combined efforts of the community and science can some day hope solve the mystery of multiple sclerosis."[25] Howard Rusk wrote in the *American Mercury* in 1947 that "for the man on the street it <MS> is merely a strange and unfamiliar medical term."[26] In short, the NMSS sought to "make the public realize by a broad campaign of education that this is not a rare or mysterious disease."[27]

To do this the NMSS fed stories and press releases to science writers and journalists at popular magazines such as: *Time*, *Newsweek*, *Saturday Evening Post*, *Look*, *Coronet*, *Parent's Magazine*, *Survey*, *Cosmopolitan*, *American Mercury*, *Reader's Digest*, *Today's Health*, *Science Newsletter*, *Science Digest*, *Business Week* and at major newspapers like the *New York Times*.[28] The successful placement of articles in popular magazines did not depend on luck. Raymond Moley, editor of *Newsweek*, served on the Board of Directors of the NMSS and Howard A. Rusk, a contributing medical editor at the *New York Times,* served on the Medical Advisory Board of the NMSS.[29] These contacts gave the NMSS significant access to popular journals.

The NMSS was able to garner more media attention and dollars by enlisting the rich, famous, and powerful in its fund-raising and organizational campaigns.[30] Senator

Charles Tobey of New Hampshire became active on behalf of the NMSS in 1948 and 1949 because his daughter, Mrs. Louise Tobey Dean, was a victim of the disease.[31] Henry Kaiser, Jr., also a victim of MS, campaigned on behalf of the NMSS in 1949.[32] The NMSS enlisted Mrs. Lou Gehrig, widow of the former Yankee baseball star, in its fund-raising drives beginning in 1949.[33] Ralph I. Straus, a director of R.H. Macy & Company, helped lead a fund-raising campaign in 1950 as did Mrs. John D. Rockefeller in 1952.[34] In 1953 the NMSS "announced the election of Edward Locke Williams as president of the Society and Oliver E. Buckley as Chairman of the Society's Board of Directors." Williams was a Long Island attorney and former president of the Insurance Executives Association. Buckley was former chairman of the Bell Telephone Laboratories.[35] Also in 1953, "with an assist from actress Shirley Temple, whose brother George has M.S., . . . the Los Angeles chapter staged a successful fund-raising telethon."[36] Mrs. Dwight D. Eisenhower agreed to be the "honorary chairman of the 1954 appeal of the" NMSS for funds and she was honorary chairman of the 1957 "Hope Chest" campaign.[37] In 1954 Ralph C. Block, Vice President of the Bank of New York, became President of the NMSS.[38] In 1955 Robert W. Sarnoff, Executive Vice President of the National Broadcasting Company, volunteered to chair NMSS a fund-raising drive.[39] In 1956 the actress Grace Kelly was chairman of women's activities section of the NMSS fund-raising drive.[40] In 1957 the NMSS appointed retired Vice Admiral H.R. Thurber as national chairman of its 'Hope Chest' campaign.[41] In 1958 Senator John F. Kennedy of Massachusetts headed the "fund-raising appeal" of the NMSS.[42] In 1959 Alfred N. Steele, Chairman of the Board of the Pepsi-Cola Company, agreed to chair the 1959 fund-raising campaign of the NMSS. His wife, the actress Joan Crawford, had served as chairman of the women's activities in the 1957 and 1958 drives of the NMSS.[43] In April 1959 Steele died and Crawford took over as chairman of the 1959 NMSS fund drive.[44] These prominent people attracted significant

media attention and their elite status also aided in fund-raising.

Grass-roots MS activists waged the public awareness and fund-raising campaigns at the local level as well. For example, in Washington, D.C. the local NMSS chapter asked Surgeon General Scheele to speak at a Shriner's luncheon "to inform leading citizens of the community . . . about multiple sclerosis." This meeting was "timed to coincide with other events connected with the M.S. campaign, such as the declaration of Multiple Sclerosis Week by the Commissioners of the District of Columbia."[45] The Washington, D.C. Chapter of the NMSS held a benefit dance "in the Terrace Room of the Shoreham Hotel on March 6, 1953" which "netted . . . approximately $2,500." The D.C. radio and TV stations gave "frequent spot announcements for M.S." Dr. Walter Freeman "was on WTOP TV from 7 to 7:15 the evening of March 17th " speaking about MS. The local chapter placed "car cards telling the M.S. story" in 400 buses and streetcars. "The March 2, 1953 issue of the 'Transit News' carried an article on Multiple Sclerosis . . ." The General Services Administration of the federal government distributed "M.S. cannisters <sic> in the government cafeterias for M.S. Week." The Washington Society of the Blind cooperated with the local MS chapter by placing M.S. cannisters <sic> "on the blind stands throughout the Washington area." The public libraries in D.C. put up MS posters and several D.C. hotels permitted the MS Society to set up public information tables in their lobbies.[46]

In New York, Governor Dewey proclaimed April 5-11, 1953 as "Multiple Sclerosis Week and asked New Yorkers to help in the fight against one of the leading disabling diseases."[47] The City of New York declared April 4-27, 1954, "MS Week," in conjunction with an NMSS fund drive.[48]

Turning now to the content of these local and national multi-media campaigns, one sees that the NMSS waged its campaign for public awareness partly by comparing the

incidence and consequences of MS with other diseases. In 1947 Marshall Hornblower, Chairman of the NMSS, writing in the *New York Times* , pointed out that "at a time when so much publicity is being given to cancer and infantile paralysis campaigns, it is important that new and unheralded organizations for pushing back other frontiers of medicine not be crowded off the pages of the press."[49] Six years later, in a letter to Surgeon General Leonard Scheele, Marshall Hornblower still perceived competition with other diseases for attention and dollars as a key element of the NMSS crusade. He wrote: "as you know, multiple sclerosis, despite its prevalence, is comparatively unknown to the general public. One of our big jobs in this area is to convince people who are already well aware of poliomyelitis, cancer, heart, etc. that multiple sclerosis is also a critically serious health problem which has been, comparatively speaking, neglected."[50]

One way the NMSS attempted to overcome public ignorance about MS was by comparing MS with polio, perhaps the most high-profile malady of the late 1940s and 1950s before the Salk vaccine.[51] *Time* magazine reported in October 1946 that "neurologists estimate that multiple sclerosis is more prevalent than infantile paralysis."[52] The *Science News Letter* went farther arguing that "the disease is believed to be more than twice as common as infantile paralysis . . ."[53] The professional *American Journal of Public Health* was more reticent than popular publications stating in 1946 that "it is thought possible that sclerosis victims may out number infantile paralysis sufferers."[54] Director of the New York Neurological Institute and Columbia University Professor and first Chair of the NMSS Medical Advisory Board, Tracy Putnam, speaking before a dinner meeting of the AARMS on February 21, 1947 announced that "in the period from 1931 to 1935 Boston City Hospital admitted twice as many people with multiple sclerosis as with infantile paralysis."[55] Howard A. Rusk announced in the *New York Times* in April 1947 that "the patients with multiple sclerosis outnumbered those with infantile paralysis by more

than two to one."[56] In October 1947 Rusk repeated the claim in the *American Mercury*.[57] The *New York Times* reiterated this claim in 1948.[58] Senator Charles Tobey deployed the alleged frequency of MS versus polio in Senate hearings in 1949.[59] Not only did the NMSS and authors report that MS was more common than polio but they argued that MS was more devastating.[60] MS patient Robert Grant, Jr. wrote in the *Saturday Evening Post* in 1953 that MS was "worse than polio, which does all its damage at one fell swoop."[61]

The NMSS attempted to get federal backing for their claims versus polio. In 1953 Alice Friedman, Director Public Relations, NMSS, asked Harold Tager, Jr., Information Officer, NINDB for support of the NMSS position being promulgated in popular publications. Friedman wrote: "I am writing to ask if you would do a little digging for me on a subject which somewhat confuses me. An article in *Newsweek*, January 14, 1952, by Dr. Pearce Bailey, estimates that 300,000 people are afflicted with MS as compared to 250,000 with the after effects of polio. (I think the 300,000 figure is conservative, considering our diagnosis problem, etc.) These figures are substantially the same as the 1952 report of the National Committee for Research in Neurological Disorders which gives figures of 300,000 for MS and the demyelinating diseases as compared with 225,000 for chronic poliomyelitis. My question is - can we say from these figures -- assuming they are correct - that MS is more prevalent than polio? This would depend on the definition of 'the after effects of polio.' But, I would like an interpretation from the National Institute."[62]

Tager responded cautiously saying that "there is a Government taboo on making comparisons of diseases, suggesting that one is more or less serious than another, and for various reasons avoidance of this kind of overt competition seems right to me. What obtains for us, however, is not necessarily so for you; but this is to say anyhow we wouldn't go on official record on statistics which are no more than educated guesses on both sides."[63] This reticence served the institutional interest of the NINDB. By

encouraging competition between diseases the NINDB encouraged more publicity for neurological conditions. This made the argument for funding research into neurological conditions and the Neurological Institute stronger.

After 1955, after the Salk vaccine, direct comparisons of MS with polio in popular magazines and journals declined dramatically. MS then emerged as the *neurological* disease which took the place of polio in public consciousness. It became what Thomas M. Rives, Medical Director of the National Foundation for Infantile Paralysis, called in 1958 the "the foremost neurological problem of our time."[64] An important pronouncement considering Rives directed the nation's largest polio foundation.

The politics of numbers as played out in popular journals held another key claim by the NMSS: that was that there were 250,000 multiple sclerotics in the United States. This assertion became a mantra in popular articles about the disease.[65] *Today's Health* reported in 1950 that "in the United States alone it is estimated that more than a quarter-million people have m.s. (as patients dub it), and the figure is probably much higher because of the difficulty of diagnosing it in the early stages."[66] In addition *Today's Health*, *Science Digest*, the *New York Times*, *Time*, *Newsweek*, and the *Science Newsletter* all reported that there were 250,000 MS sufferers in the United States from 1950 to 1957.[67] Occasionally there would be even higher claims of MS incidence. *Look* said in 1954 that there were 300,000 cases of MS in the United States.[68] In 1956 the *New York Times* reported 500,000 American MS patients;[69] but in 1957 the *New York Times* stated the number to be 300,000.[70] In 1957 *Newsweek* upped its estimate to 300,000.[71] These numbers were plastic estimations and not based on solid epidemiological evidence.

In fact epidemiological studies sponsored by the NMSS and the United States Public Health Service (USPHS) in 1948 through 1951 had come up with much lower figures. Charles C. Limburg's study had found that there were between 50,000 and

150,000 cases in the United States.[72] Dr. Leonard Kurland's survey had found an incidence of 70,000 to 80,000.[73] The NMSS and the NINDB ignored these studies which they had funded and continued to claim that there were between 250,000 and 300,000 cases of MS in the United States. Even in the same publication one could find these competing estimates. In the 1954 publication of the proceedings of the 1953 conference entitled, "The Status of Multiple Sclerosis," O.E. Buckley, Chairman of the Board of Directors of the NMSS, maintained that there were "from 200,000 to 300,000 cases of" MS in the United States. Contradictorily, Leonard T. Kurland of the NINDB and Knut B. Westland of Johns Hopkins estimated that there were "about 70,000" cases of MS in the United States.[74] Of course it was in the interest of the NINDB and the NMSS to publicly hold to the higher numbers which they could always support through justified claims about the difficulty of diagnosing MS. (See chapter two). However, considering that in 1995 the estimate of MS cases in the United States remains 250,000 to 350,000 the two organizations probably deployed inflated numbers in the 1950s given the large population increase in the USA since the 1950s.[75]

These prevalence claims did not go uncontested. In April 1954 Lawrence C. Kolb, M.D., of the Mayo Clinic in Rochester, Minnesota wrote the Medical Director of the NMSS, Harold R. Wainerdi. Kolb complained that he "was rather distressed, in reading the annual report of the National Multiple Sclerosis Society, to notice under the section dealing with prevalence the statement that 'the National Institute of Neurological Diseases and Blindness estimates that there are approximately 300,000 person in the United States today with chronic, progressive, multiple sclerosis and related demyelinating diseases.' I do not know who is responsible for giving you this figure, but it is entirely out of line with the work that was done by your own statistical committee under the directorship of Dr. Leonard Kurland who, of course, is now attached to the National Institute of Neurological

Diseases and Blindness. The figure is more like 70,000 or 80,000." [76]

Another element of the public relations campaign waged by the NMSS had to do with the cultural syntax writers used to represent MS patients in the popular literature. Authors designed their magazine articles not only to increase public awareness of the malady but to instill hope in MS sufferers. This was vital because of the despair many MS patients felt upon diagnosis. I found many cases of suicide attempts, depression, and despair in patient records.[77] One UCLA doctor described a patient's depression this way in his "Physician's Notes.": "Her depression is mostly from mistaken ideas of M.S. and her prognosis which she makes unrealistically morbid. Is currently sitting around home not working and only brooding because she felt working would make M.S. worse. Parents also are over concerned and cautious and slowly driving pt into nervous exhaustion from too much misguided <sic> attention. Have urged her to return to work and straightened out some of her misconceptions."[78] One way popular writers instilled hope was to point out that the average life span of the MS patient was only slightly less than normal. Another way writers constructed the trope of hope was to proclaim that in the absence of an absolute physiological cure healing could occur if the individual patient enacted the American myth of self-transformation.[79] To be healed of MS meant to transform oneself through individual effort, to resurrect oneself.[80]

For example, Paul de Kruif, in "The Patient is the Hero," *Reader's Digest* (1948) recounted the following tales of remission/resurrection: "in 1941, the *Washington Evening Star* carried a headline: HOPELESS CRIPPLE CONFOUNDS DOCTOR IN TRICYCLE TRAVELS. On his bed Wilford Wright had begun feebly but systematically to move his stiff, wasted muscles. At last he struggled up onto an adult's tricycle. Since then he's driven his tricycle from Florida to Nova Scotia and even to the West Coast. He is not completely rehabilitated, but his improvement is remarkable."[81] "In Cleveland, a young

woman, Betty Bard, lay paralyzed from multiple sclerosis. On her own she began giving herself weak but infinitely determined exercises. She is medically famous as an advanced case now free from incapacity." "Twenty years afflicted, five years bedfast, Mrs. Henrietta Apatta was learning to walk, alone and unsupported."[82]

The emphasis was on individual effort and self-transformation through the use of "heavy resistance exercises." These labors when carried on with devotion might "mean resurrection to active life."[83] As T*oday's Health* reported in 1950: "patients must have the will to work."[84] Commenting on her own recovery in 1950, also in *Today's Health*, Jean Griffith Benge reported that "it took character and persistence, for the amount of work to make any progress is prodigious."[85] Benge witnessed to other sufferers: "my experience brings a message of hope to parents whose children have had polio, to those afflicted with m.s. and to sufferers from other paralytic conditions."[86]

Cosmpolitan magazine featured Joan McCarthy's, "My Victory Over MS," in 1960. In the article MS patient McCarthy advised MS patients to "make your own miracle." If MS patients wanted recovery McCarthy preached that "you've got to make it happen yourself."[87] McCarthy recounted her own experience of "soul-searching." She overcame despair and hopelessness in a critical turning point in 1954 which she described in the syntax of a conversion experience. She then knew "that it was up to me to recover. Nobody could do it for me."[88] Later, MS patients asked McCarthy how she had recovered some of her lost abilities like walking and driving. She "told them it was nothing but work, and that work was everything."[89]

In 1953 the NMSS in a pamphlet entitled, "Self Help," advised that until there was a cure "the multiple sclerosis patient will fundamentally make his adjustments through his own resources--through courage, persistence and self-discipline. More specifically, the multiple sclerotic should be aware of the dangers of depending too greatly on his family,

desirable though this course may seem to be. Such dependence may aggravate the sufferer's condition; and in any case, the dependent patient rarely receives enough sympathy to satisfy him, and he becomes more and more of a burden to himself and his family. Only be maintaining a sure independence, within medical limits set by a physician, can the patient live a full creative life."[90]

Similarly, in 1956, *Coronet* magazine prefaced an article by multiple sclerotic Jane Sterling with: "This is the philosophy of a dauntless woman who found the power within herself to conquer her crippling malady."[91] Sterling taught that "self-discipline is of utmost importance if one is to cope with the disease successfully."[92]

Not only could patients transform themselves through individual effort but they could experience healing through fellowship with other multiple sclerotics. In 1948 the NMSS framed the plight of the MS sufferer this way: "the Multiple Sclerosis victim has been peculiarly isolated . . . in the Biblical phrase, 'a man sitting in darkness.'"[93] To overcome this isolation the NMSS encouraged patients to engage in collective self-help. By 1953 the NMSS reported that many local chapters had "established patients' clubs and some patients' clubs have been founded locally without affiliation with any organization. Such clubs, apart from the therapeutic facilities they may afford provide a useful social outlet for multiple sclerosis patients; they afford the ease of friendship and the common, sympathetic understanding of those who share the same difficulties. Meeting together, multiple sclerosis patients also have the opportunity of sharing information about hobbies and business or employment opportunities."[94]

Other collective experiences could be found in clinics, funded by the NMSS, devoted to multiple sclerosis research and treatment in key cities around the country. In 1948 the NMSS funded MS clinics in Boston at Beth Israel Hospital, Boston State Hospital, and Massachusetts General Hospital. Also in 1948 the NMSS funded MS

Clinics at Tulane University School of Medicine in New Orleans, Cedars of Lebanon Hospital in Los Angeles, the New York University School of Medicine and Montefiore Hospital in New York City, and at the Albany Hospital in Albany, New York."[95] In 1954 the NMSS and its Washington Chapter sponsored a new clinic at George Washington University Hospital in Washington, D.C.[96] The stated purpose of these clinics was to do research. However, these clinics, along with the patient clubs, also served as sites around which a movement culture could develop and sites where ongoing fund-raising activities could occur. As one lay director of the Washington, D.C. chapter of the NMSS put it in 1953: "something concrete must be offered to the patients . . . The chapters were authorized to keep 60% of their funds in the local area . . . this was a wise plan because without an information center, a clinic and efforts at rehabilitation, it would not be possible to raise any money for research." Pearce Bailey concurred saying that "unless a clinic is set up the Chapter will not survive. Patients need a program." One physician commented that the clinics acted "as psycho-therapy. The patients are happier if they have somewhere to go."[97]

In addition to metaphors of self-transformation and collective self-help, patients and lay activists also deployed military metaphors in framing MS in popular journals. One patient wrote in 1950 that "muscle reeducation like Pavaroff's is a calculated campaign, plotted a precisely as any of the world's great battles."[98] Another multiple sclerotic described his disease in 1954 as a "phantom sniper" and that "mystery" was its "Iron Curtain."[99] *Newsweek* editor Raymond Moley in, "Weapons Against a Pitiless Enemy," compared MS to a "guerrilla" attack; "MS attacks here while it retreats there . . . "[100] While it might be tempting to attribute this military framing of MS as due to World War II or the Cold War, military metaphors were not an unusual formulation for diseases nor were they unique to the 1940s and 1950s. Nevertheless, the military jargon was an important element

of the popular construction of the disease and the campaign against it. The martial language also helped to create a sense of mission in the popular crusade.

Healing, then, as the NMSS represented it, was to participate in a crusade against the disease itself. In other words to struggle against disability and death in a movement culture animated mainly by the American myth of self-transformation and collective self-help. Moreover, in terms of an ultimate cure, the patients' participation was key as well; according to the NMSS "only the combined efforts of the community and science can some day hope to solve the mystery of multiple sclerosis."[101]

IV The Second Front: The National Multiple Sclerosis Society and the National Institute for Neurological Diseases and Blindness

The second major push of the NMSS in the late 1940s and 1950s consisted of lobbying the federal government to fund research on MS as well. The initial stages of this campaign show the extent to which the lobbying effort was embedded in a new postwar cultural consensus concerning the relationship between disease, medical research, and the federal government.[102] The federal government was also enthusiastic about funding medical research because of the successes which flowed from the funding of military related medical research in World War II. With the Cold War afoot, funding medicine became part of the national security apparatus.[103] This interest of the national security state in issues of biomedicine spilled over into multiple sclerosis research as well. In August 1959 "Dr. Clifton Himmelsbach and Dr. DeLong from the Central Intelligence Agency" contacted NINDB physician Richard Masland because the CIA doctors had a "serum from a patient treated in Russia for multiple sclerosis with a Russian anti-multiple sclerosis vaccine. They are anxious for arrangements to be made for this serum to be tested."[104]

However, it was not just state interest that accounted for the postwar research explosion. The "wartime parade of miracle drugs," especially penicillin, led American

society to call "for more and more medical research" for which it was prepared "contribute handsomely" as medical workers at the time knew.[105] Cornelius H. Traeger, M.D., expressed the new faith in science when in 1949 he preached that "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."[106] As one patient put it in 1954: "I know that, in this age of atomic energy, antibiotics and radioactive isotopes, a cure for my trouble is around the scientific corner."[107] This vaulting cultural faith in medical science led to a new consensus that, as Mayor Wagner of New York City expressed it in 1956, "government must play its role, in sponsoring medical research."[108]

The federal government had been involved in medical research before World War II; however, the amount of money the government spent on research before and after 1942 were of far different magnitudes.[109] In 1887 the Marine Hospital Service set up the Hygienic Laboratory to do bacteriological studies to help control epidemics. The Biologics Control Act of 1902 authorized the Hygienic Laboratory to test biologicals and to expand into zoology, chemistry, and pharmacology; yet, its annual budget was less than $50,000. In 1912 Congress authorized the Public Health Service (USPHS), formerly the Marine Hospital Service, to study chronic and infectious diseases.[110] Through the 1920s the USPHS budget was still only about $300,000 per year.[111] In 1930 Congress authorized the conversion of the Hygienic Laboratory of the USPHS into the National Institute of Health and the spending of $750,000 for this purpose; however, in the 1930s Congress never appropriated full funding for the NIH because of the Great Depression. In 1937 Congress created the National Cancer Institute and appropriated $400,000 for its first year; however, in 1945 The NCI budget was still only $500,000 per year.[112] The low NCI budget notwithstanding, the federal government vastly expanded medical research in World

War II. The Committee on Medical Research in the Office of Scientific Research and Development spent $15,000,000 during the war. The momentum for federal funding of medical research continued as the government closed the OSRD/CMR at the end of the war and transferred remaining funds to the NIH. The NIH budget quickly expanded; its research budget went from $180,000 in 1945 to $4,000,000 in 1947. In 1945 Congress appropriated $700,000 total for the NIH. In 1948 the government expanded NIH spending to $29,000,000 and by 1955 the NIH budget was $98,000,000. By 1965 the figure had reached $436,000,000.[113]

The federal government established other research institutes through the 1940s with these monies. By 1950 the NIH included the National Cancer Institute, Experimental Biology and Medicine Institute, Microbiological Institute, National Institute on Mental Health, National Heart Institute, and the National Institute for Dental Research.[114] In this cultural and political context the MS Society lobbied the federal government to start a Multiple Sclerosis Research Institute like the other dedicated institutes. The following comments show the arguments that the MS activists and their allies used to get the federal government involved in MS research but they also show the extent to which a change had occurred in American culture, i.e. disease and medical research were legitimate and necessary activities of the federal government. Solving disease had come to be seen as just another public works project for which many people wrote their congresspersons.[115] For example, one patient wrote in 1948: "Dear Senator Wherry: . . . in your position in the U.S. Senate, it occurred to me that you might see the opportunity of the establishment <sic> of laboratories or hospitals which could be devoted to the effort to establish the cause of this most baffling and insidious disease, about which at this time practically nothing is known."[116] Here is another example of how health projects came to be viewed like bridges, roads, dams and other public works projects. In 1948 U.S. Representative

Mike Mansfield of Montana forwarded a letter from a constituent to Surgeon General Leonard Scheele. Mansfield wrote: "herewith is letter which I have received from Miss Jane Sullivan relaitve <sic> to sclerosis. It is the intention of a group in Butte to carry on some reserach on this disease and they were wondering whether or not any research could be done at the Laboratory at Hamilton, Montana."[117]

In addition to grass-roots letter writing, the NMSS activists initiated legislative action in the Senate. On May 10, 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.* Republican Senator Charles Tobey of New Hampshire, whose daughter had MS, sponsored the bill. Tobey declared that "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."[118] Not only had studying disease become a legitimate concern of the federal government it had become a moral imperative.

Ralph I. Straus, President of the NMSS, stated succinctly the postwar acceptance of the significantly increased role of the federal government in medical research: "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."[119]

Cornelius H. Traeger 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."[120] Tracy Putnam concurred saying that "it is my personal belief that the time has come when we must turn to the State and Federal Governments for aid in the struggle against a disease such as multiple sclerosis, which carries such tremendous misery with it and economic loss."[121]

One Illinois citizen echoed the doctors' sentiments in a letter to Senator Tobey: "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."[122]

In these hearings and elsewhere, MS activists claimed that the NIH in particular and the medical profession in general had ignored their disease even though the pace of federal funding for medical research was increasing. In 1946 *The New York Times* hinted at negligence on the part of medical workers: "this ignorance of a disease which is a social problem is no credit to science . . . The Association <AARMS forerunner to NMSS> has engaged in work which should have been undertaken systematically long ago, and which deserves all the philanthropic support that it can enlist."[123]

Testimony at the 1949 Senate hearing demonstrated this sentiment of neglect as well: Senator Tobey complained that "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 agreed saying "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." Ralph I. Straus, President of the NMSS, concurred testifying 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."[124]

These charges were not accurate as neuroscientist Tracy Putnam pointed out in the 1949 Senate hearing. In fact American neurologists had been investigating multiple sclerosis since the late nineteenth century and had explored numerous hypotheses concerning the etiology of MS. The Commonwealth Fund financed MS studies at the New York Neurological Institute from the 1920s to the 1940s. (See chapter four.) Physicians working for the military during World War I had conducted the first proto-epidemiological study of MS in the United States based on the famous study, "Defects in Drafted Men."[125] What *was* accurate was that the Public Health Service and the NIH had virtually ignored MS and, in fact, had mostly ignored neurological questions altogether.

In 1947 the study sections of the USPHS research grants programs included: antibiotics and bacteriology, biochemistry and nutrition, cardiovascular, dental, gerontology, hematology, malaria, metabolism and endocrinology, pathology, pharmacology, physiology, public health methods, radiobiology, sanitation, surgery, syphilis, tropical diseases, tuberculosis, and virus and rickettsial diseases.[126] There was no neurology study section.

In a 1948 letter to Cornelius Traeger, of the NMSS, David E. Price, Chief, Division of Research Grants and Fellowships, USPHS, discussed the lack of interest of

the USPHS in multiple sclerosis research: " . . . the Public Health Service has received very few applications for research projects relating to the demyelinating diseases. I believe I am correct in saying that to date we have not received any requests for grants that relate directly to multiple sclerosis . . ."[127] There still was no neurology study section within the USPHS through 1950.[128] However, the USPHS through the NIMH had begun an epidemiological study of MS in 1947 in cooperation with the NMSS.[129]

Despite these pleas and the fact that the NIH was basically not much involved in neurological research, the NIH opposed the formation of a separate institute for multiple sclerosis because of the administrative burden of a separate institute for one particular disease. They also opposed Tobey's bill because it would set a bad precedent if every disease required its own institute. As a result the NMSS changed its tactics and lobbied for the founding of a neurological institute with funding for multiple sclerosis research.[130]

These lobbying efforts of the MS activists succeeded. On August 15, 1950 President Truman signed Public Law 692 which authorized the Surgeon General of the USPHS to set up the National Institute for Neurological Diseases and Blindness (NINDB) and the National Institute for Rheumatism and Metabolic Diseases. NIH officials began work to organize the institute; however, Congress did not appropriate funds for the institute until 1952.[131]

With the founding of the NINDB the NIH's interest in multiple sclerosis increased significantly. Table 8 shows the amount of money the NINDB spent on MS in selected years. The total for 1955 represents the amount of money appropriated through August.[132] Altogether, by August of 1955 the NMSS had persuaded the federal government to spend $1,035,166 on MS research.[133] These dollars represented NINDB support of seventeen research projects in 1954. I have incomplete data for 1955 but by August of 1955 the NINDB was supporting eighteen projects related to MS for the year.[134] By 1955 the

NMSS had spent $1,355,642 on research which went meant that between 1946 and 1955 the two organizations had been pumped $2,390,808 into MS research in the United States.[135]

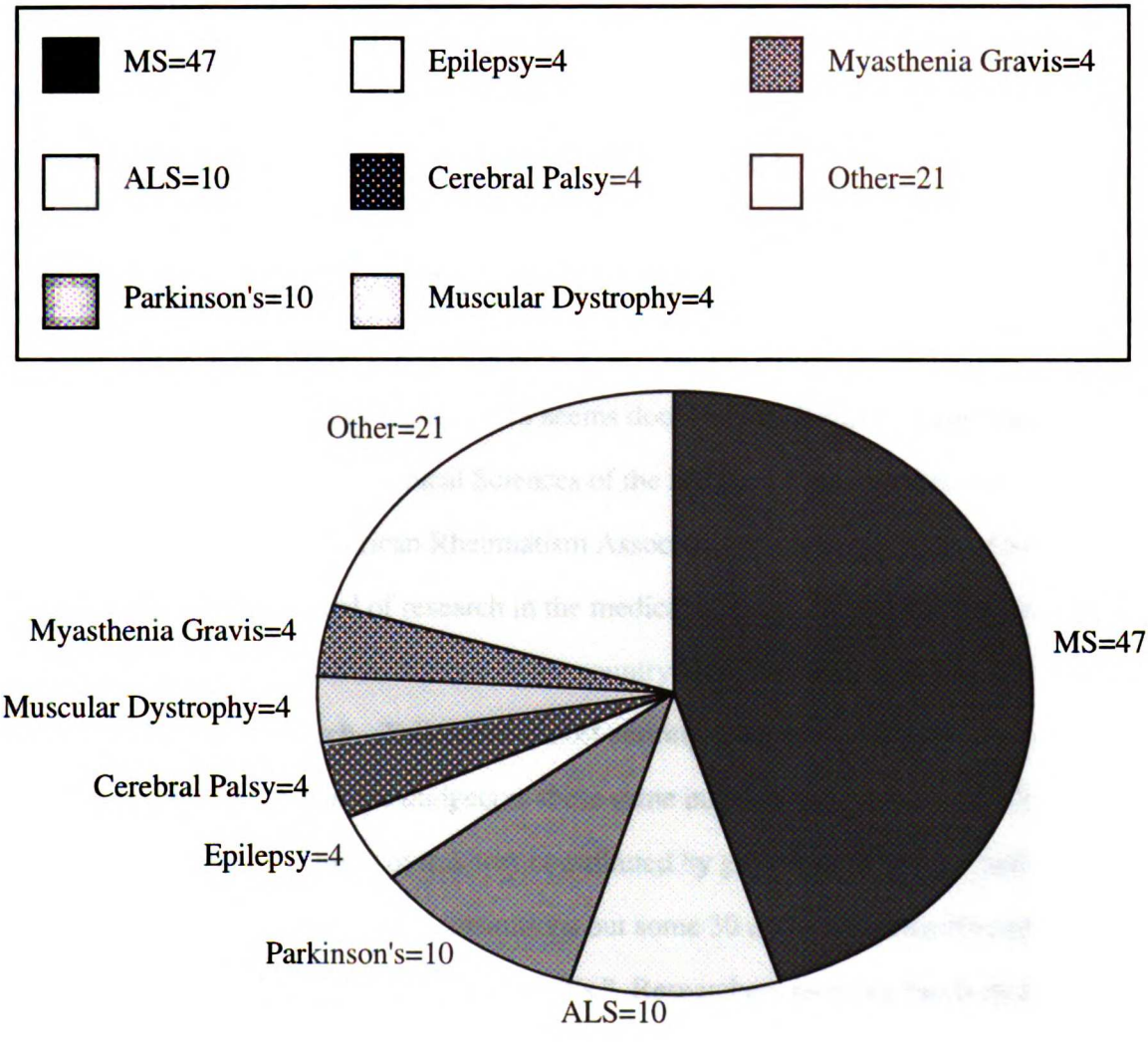
The NINDB's mandate was to study epilepsy, cerebral palsy, multiple sclerosis, blindness and other neurological diseases. However, the MS patients seemed to have been the most active partisans of a particular disease in lobbying the NINDB. See Table 9 which compares requests for admission to the NINDB Clinical Center, an eight bed facility still in the planning stages, from June 10, 1952 to July 10, 1953.[136]

The NMSS had incorporated Amyotrophic Lateral Sclerosis (ALS) within its purview which meant the scleroses together accounted for fifty-five percent of all requests to the Clinical Center during this time period. In 1952 the NINDB spent as a percentage of the total money for research, for the two largest categories, 11.9 percent for epilepsy and 11.5 percent for multiple sclerosis.[137] This represented a significant accomplishment for the NMSS activists but it was not just the pressure of the NMSS which made this spending possible. It was in the interests of neurologists as a specialty and the NINDB as an institution to ensure close cooperation between the NMSS and the NINDB. Neurologists successfully captured and channeled the lay pressure of MS activists towards the goal of expanding their specialization.

American neurologists needed the lay activists to expand their specialty. The following 1955 assessment of Roswell B. Perkins, Acting Secretary of the Department of Health Education and Welfare, shows how the expansion of MS funding facilitated an expansion in neurology training: "one of the primary drawbacks to an accelerated research attack on multiple sclerosis is the lack of trained investigators in the neurological diseases, a shortage which the Institute is endeavoring to overcome . . . There is undoubtedly some room for expansion of studies in multiple sclerosis. Such expansion, however, must

Table 9

Requests for Admission to NINDB Clinical Center June 10, 1952 to July 10, 1953



depend on the availability of more trained research manpower."[138] The lack of trained neurological researchers still remained a problem in 1960.[139]

It would be wrong however to understand this as a situation where one private organization, the NMSS, pressured a public institution, the NINDB. An analysis of the interaction between these two groups shows that they virtually acted as one organization. In fact there was a very blurry boundary between what could be considered public and what was private between these two groups. Through a system of interlocking directorates and close cooperation the NMSS and the NINDB established what was in effect a single structure ruling neurological research at least with regard to MS.

That this model was unique to MS seems doubtful. R. Keith Cannan, Vice Chairman of the Division of Medical Sciences of the National Research Council, in a speech delivered to the American Rheumatism Association on May 28, 1953, described theevolution of a new model of research in the medical sciences. Cannan remembered that "prior to 1942, the medical institutions of the country were spending less than 5 million dollars annually on research. This came almost entirely from private sources. Government contributed little. Last year the budgets of these same institutions approached 100 million dollars-- and about two-thirds of this was contributed by government. About half of the latter was expended within federal institutions but some 30 million was distributed to academic centers in the form of research grants." Researchers received funds from the federal government and also from "national societies such and the Arthritis and Rheumatism Foundation." Cannan lamented that these research dollars tended to "arrive elaborately packaged in restrictive conditions. And the responsibility of trusteeship has passed from the university to committees in Washington . . Control has passed, in large measure, out of the hands of the universities into those of government and private agencies."[140]

Another indication that the structure of the relationship between the NMSS and the NINDB was not unique to MS comes from minutes of the National Advisory Neurological Diseases and Blindness Council, from November 16, 1950 which read: "it was brought out that there is close coordination between the various national and private medical associations and organizations and the National Institutes of Health. Individual members of the councils have close relationships with the large foundations, and they are able to carry back to them the activities of the councils, and, in turn, are able to bring to the councils their knowledge of the private organizations . . . A new pattern is developing in research in which government funds are playing an increasingly important role, and this support by government has stimulated support elsewhere . . . the one complements the other."[141]

How private foundations and the NIH supported each other, at least in MS research, was through a system of interlocking directorates which partly determined the structure of the relationship between the NMSS and the NINDB. As Surgeon General Leonard A. Scheele put it in 1953: "the relations between the Public Health Service and the National Multiple Sclerosis Society have always been very close."[142] What is surprising is how close they were and the extent to which the agencies virtually acted as one organization at least with respect to medical research and public education. Members of the NMSS Medical Advisory Board served on the NINBD's National Advisory Neurological Diseases and Blindness Council.[143] And officials at the NINDB served on the Medical Advisory Board of the NMSS. For example, in 1951 Cornelius H. Traeger served as Medical Director of the NMSS and a member of the NINDB Council. Pearce Bailey was Director of the NINDB and served on the NMSS Board and the NINDB Council as did Dr. H. Houston Merritt and Dr. S. Bernard Wortis. The NMSS had a strong presence on the NINDB Advisory Council. In 1951 four of the thirteen members of the NINDB Council

were on the NMSS Medical Advisory Board. In addition, Dr. R. H. Felix and Dr. John C. Eberhart, represented both the NMSS and the NIH at the June 1951 meeting of the NINDB Council. In 1955 Cornelius H. Traeger served as Medical Director of the NMSS and he also was Chairman of the Council Committee on Program Planning of the NINDB Advisory Council.[144]

In 1951 the NMSS appointed Frederick L. Stone, Chief of Extramural Programs at the NINDB to the NMSS's Medical Advisory Board.[145] Stone enthusiastically served on the board saying "I will most certainly be up to the meeting of the Medical Advisory Board and appreciate the privilege of serving upon it, and will do my best to be of maximum service in any capacity whatsoever."[146] Stone worked actively to "forward" the "mutual program interests" of the NMSS and the NINDB.[147] In December 1951 Stone wrote Traeger, "concerning the functioning of the National Advisory Neurological Disease and Blindness Council" specifically the "method of reviewing applications" for research. Stone, the federal official, asked Traeger, the private director, if he was "satisfied with the present system, and if it can be changed to meet with your approval, within our present procedural framework."[148]

Conversely, the private NMSS asked the public Stone to approve NMSS research grants.[149] Stone also served on the smaller executive committee of the NMSS Medical Advisory Board which could authorize research expenditures of one thousand dollars or less without the full board's approval. Traeger asked Stone to approve a research request in April 1952. The project had "to do with the analysis of cerebrospinal fluid for amino acids . . ." Drs. Burnham S. Walker and Dr. Joseph Foley were to obtain the spinal fluids from Boston City Hospital. Stone approved the request by phone on April 11, 1952.[150] Then, in 1955 Stone quit the NINDB and went to work for the NMSS becoming director of the NMSS Medical and Scientific department.[151]

Pearce Bailey, Director of the NINDB, also served on the Medical Advisory Board of the NMSS.[152] It was more than just a *pro forma* affiliation. Bailey depended on the NMSS for the planning of the NINDB. In 1951 Bailey wrote Cornelius Traeger, NMSS Medical Director, asking for help saying that "one of the first tasks in the activation of the National Institute of Neurological Diseases and Blindness is to assemble some factual data to consolidate its program, or any expansion thereof. It is important that such data be obtained as early as possible, even at the expense of its absolute accuracy."[153] In 1954 Bailey sent Traeger "the fact sheet outlining the general program needs of the National Institute of Neurological Diseases and Blindness for fiscal year 1954."[154]

Cornelius Traeger asked Bailey to approve Montreal neurologist "Roy L. Swank as a liaison member of the Medical Advisory Board of the National Multiple Sclerosis Society" in April 1952. Bailey responded that he "heartily" approved the nomination.[155] The NMSS routinely asked Bailey to approve other nominations to the Board, at least through 1955 when Bailey was serving on the Nominating Committee of the Medical Advisory Board.[156]

Bailey met privately with Sylvia Lawry, Founder/Director of the NMSS, and Traeger in New York City in February 1952 to discuss common agendas and strategies and to solve their, as Bailey put it, "mutual problems."[157] In 1953 the NMSS sponsored a conference on MS in New York which Pearce Bailey chaired.[158] Later that year, Ralph C. Glock, President of the NMSS, wrote Bailey asking him if he was "able to suggest one or more candidates for the position of Medical Director of this Society . . ." Bailey declined to suggest anyone though it is not clear why. It might have been politically imprudent for him to do so.[159] Nevertheless, remembering from above, in 1955 Frederick L. Stone, a NINDB official, became director of NMSS Medical and Scientific Department.[160]

The structure of the relationship between the NMSS and the NINDB was not

dependent on particular personalities. For example, in 1960, after becoming the new director of the NINDB in December 1959, replacing Pearce Bailey, Richard L. Masland also joined NMSS Medical Advisory Board.[161] In June 1960 Thomas L. Willmon was the new Medical and Research Director at the NMSS. Willmon wrote to Masland for his advice: "referring to your letter of 11 June 1960, the MS Society is providing 'a continuing stimulus for further efforts' directed toward development of protocol for evaluation of therapy in multiple sclerosis through conversation, etc. Is there any other action you would like taken?"[162]

One consequence of this system of interlocking directorates with its politics of reciprocal patronage was that in practice there was a very blurry boundary between the public and private spheres. For example from 1948 to 1951 the NMSS and USPHS co-funded an epidemiological study of MS under the direction of Lawrence C. Kolb of the USPHS. When the studies came back with lower estimates for the incidence of MS than the sponsors had hoped for, it was in the institutional interests of the NMSS and the NINDB to ignore the results. From 1948 onward the NMSS, first with the National Institute of Mental Health then with the NINDB, closely coordinated research projects to avoid duplication.[163] This meant that in strategic and tactical planning the private NMSS and the public NINDB acted as virtually a single entity. This cooperation extended into other spheres; for example, many MS patients or their family members wrote the NINDB asking for advice about treatments and physicians. The NINDB directed the inquiries to the NMSS.[164]

There were some legal boundaries that defined a boundary between the NMSS and the NINDB but these were overcome as well. For example, in 1953 the NINDB wanted to buy 10,000 copies of a pamphlet from the NMSS but legal questions arose whether it was permissible for the NINDB to do so. The NMSS and the NINDB had jointly planned the

pamphlet in 1952.[165] Pearce Bailey remembered that "early in 1952, representatives of the Institute and of this Society began discussion of the possibility of a pamphlet that would meet both the needs of the Society and the responsibilities of the Institute. It was apparent <sic> that two publications should be avoided not only to eliminate waste, but also to avoid the impression that there were differences of opinion between public and private agencies concerned and to avoid almost certain discrepancies in the scientific information which would be made available . . . the decision reached jointly was that the Society rather than the Institute should produce, publish and distribute the pamphlet." Government lawyers approved the purchase of 10,000 copies. In 1955 the NINDB purchased 5,000 copies of a different pamphlet from the NMSS.[166]

The NINDB/NMSS circumvented another government policy which was intended to maintain the public/private boundary. In 1955 the NINDB was having problems "concerning qualified candidates for appointment as Fellows and Scholars, for the support of whom" the NINDB might not have funds. Harold R. Wainerdi, Medical Director, NMSS, proposed to Pearce Bailey that "if you would like to send us the names of those applicants whose interests lie in our area and who would probably merit consideration, I could write them a diplomatic letter, stating that I have been informed of their interest in such support, and am therefore, calling our program to their attention . . . This might result in our having a somewhat better field of candidates from which the Fellowship Committee would make its choice."[167] Bailey "explored this problem with the Division of Research Grants of the" NIH. Unfortunately, it was NIH policy "because of the personal nature of an application for a fellowship not to divulge the names of applicants."[168] Wainerdi wrote back saying that "if you are not able to release the names of your applicants, would it be possible to advise those applicants who for some reason cannot be supported and who in your judgment have merit of our interest in this matter so that they might be directed toward

us?"[169] Bailey responded that yes he could do that and he went farther saying that "Dr. Seger suggested that you might wish to know of applicants for clinical traineeships who are interested in multiple sclerosis. The number of candidates that we have for this latter type of award is always far above the number we can support from our limited appropriation."[170]

Another consequence of the interlocking directorates, the politics of reciprocal patronage, and the blurry boundary between public and private spheres was that the NMSS/NINDB served a disciplinary function with regards to MS and neurological research. The NMSS/NINDB coordinated efforts to put down local, independent investigators and one local, independent MS voluntary group in order to establish tight control over MS research in the United States. In 1952 Dr. Emanuel M. Abrahamson of New York City came out with a treatment for MS which he had developed while not under the control of the NMSS/NINDB. Abrahamson contended that "all multiple sclerosis patients have hyperinsulinism and that he" had "been able to produce excellent therapeutic results by a diet . . . calculated to correct the hypoglycemia." The NMSS/NINDB coordinated their public response to the incident.[171]

In 1954 Drs. Milo G. Meyer, Alan Johnston and Arthur F. Coca published an article which claimed that the elimination of allergens could cure or improve MS.[172] The NMSS/NINDB also coordinated their public response to this independent group.[173]

In another example, in 1952 a turf battle broke out between the NMSS and an unaffiliated local MS voluntary group in Chicago allied with independent researchers at Northwestern University. The NINDB cooperated with the NMSS in overcoming this site of independent and local research and voluntary organization. Sylvia Lawry, Founder/Director of the NMSS, described the rogue group in an internal NMSS memorandum forwarded to Pearce Bailey at the NINDB: "The program of the M.S.

Foundation consists entirely in the financing of a multiple sclerosis research program at Northwestern University for a five-year period at an annual budget of $25,000, under the direction of Dr. Lewis J. Pollock . . . there is no Medical Advisory Board except for personnel of Northwestern University. Therefore, no objective review of this project by a medical body has been effected . . . Our negotiations with the M.S. Foundation, primarily through Mr. Francis Abeles, its past President and now Honorary President, have been unsuccessful due mainly to our reluctance to concede to their request that they be given equal representation on our Board of Directors . . . In addition, the Chicago Foundation had expressed the desire to spend its funds for research as it sees fit, without submitting its research program to our Medical Advisory Board for approval . . . "[174]

Alarmed at the Chicago group's reluctance to merge with the NMSS, Harold R. Wainerdi, Acting Medical Director, NMSS, wrote Pearce Bailey warning that "this group recently has expanded its name to give a national implication so that where we have been accustomed to refer to it as 'the Chicago Group' or 'the Multiple Sclerosis Foundation,' it is now designated as the Multiple Sclerosis Foundation of America . . . " Wainerdi found the Chicago group "somewhat unsophisticated in their truculent attitude toward working with a larger group such as ourselves, whose interest is obviously selfless and who have developed extremely intricate controls to insure the quality of work performed under our aegis." The problem was one of local control. Wainerdi commented that "there is an extensive and intricate relationship with Northwestern University about which I believe we still do not have complete details, but the influence of that Institution in the city of Chicago appears to be so dominant that few medical persons in that community were, until recently, willing to work independently." Apparently the Chicago group did not want to "surrender hegemony to an Eastern organization . . ." The NMSS/NINDB appointed University Wisconsin Professor Hans H. Reese to establish an orthodox MS society in Chicago.[175]

In effect, the NMSS/NINDB disciplined the boundaries of what was considered

legitimate MS research and effectively nationalized that control. The result was a new national model of finance and control of medical research which overcame the previous structure of local, independent, ad hoc, private foundation based research that had dominated the United States in the 1920s and 1930s. The evidence suggests that this was perhaps not unique to MS but a consequence of the *de facto* merging of the NINDB with private voluntary organizations in the early 1950s.[176]

This system of interlocking directorates extended throughout American neurology in the 1950s. In 1952 the American Neurological Association elected Dr. Hans H. Reese as its President and the NMSS named him Chairman of the Medical Advisory Board.[177] In 1955 the Neurology Subcommittee of Veterans' Administration Advisory Committee included the Chairman Dr. H. Houston Merritt, Dr. Pearce Bailey, and Dr. S. Bernard Wortis. All of these men served on the NMSS Medical Advisory Board and the NINDB Advisory Council as well.[178]

An example of the coordination of the various bodies can be seen in the behind-the-scenes maneuvering of Dr. Leonard T. Kurland to get his research project funded and approved. In a 1952 letter to Pearce Bailey, NINDB Director, Kurland proposed that

> in accordance with decisions reached at our meeting with Dr. Price and Dr. Kety, the American Academy of Neurology and/or the American Neurological Association will arrange (by some method you will devise) to form a committee to study the frequency and distribution of the neurological disorders. This committee, after considering the problem and recognizing the difficulty and expense involved, will invite the U.S. Public Health Service and various interested voluntary agencies to participate in the project. Upon receiving the request for assistance from the committee, you might then make available the services of the epidemiologist whom the committee could appoint as field director of the project. The epidemiologist would be asked to prepare a detailed plan for the study. . . The epidemiologist, as agent of the committee, will apply for supplementary funds from the voluntary agencies for the six university grants.[179]

Bailey then forwarded Kurland's research proposal to Dr. Augustus S. Rose, Professor of Neurology at UCLA and chairman of the research committee of the American Academy of Neurology. Bailey expected Rose to comment on "the possible role the Academy might play in the project."[180]

This example shows how neurologists as a specialty effectively captured the research dollars flowing from NINDB and the voluntary societies and put them in what was, in effect, a single pot. The NINDB/voluntary society oligopoly also allowed American neurologists to tighten the hierarchies of their profession and to overcome localism in research-not however in treatment at least in the 1950s. Dr. H. Houston Merritt commented on the National Committee for Research in Neurological Disorders in 1958: "this Committee is composed of two of our largest neurological societies and the voluntary health organizations vitally concerned with the impact of neurological disorders upon our society . . . the committee was organized in 1952 to assist the Director of the National Institute of Neurological Diseases and Blindness in blueprinting a national research program in neurological and sensory disorders. It serves to coordinate research programs in these areas and helps to prevent the splintering of such research from its central core. Each year it studies carefully the research and training fund needs of the Institute and makes a budgetary estimate of those needs . . ." What was key to the formation of this nationalized structure of neurology research was the *de facto* merging of the voluntary societies and the NINDB into one organization.[181]

V Conclusion

In conclusion, MS emerged as a popular crusade and a research priority in American neurology and the federal government in the late 1940s and 1950s because patients and their partisans collectively organized and pressured medicine, the government, and society to fund research on their disease. However, part of the reason for their success

was the favorable political and cultural climate of the time for the expansion of biomedical research generally. Another reason for this success was that American neurologists quickly recognized the opportunity increased research dollars meant for their specialization and through a system of interlocking directorates neurologists as a specialty colonized and controlled the research dollars of the NMSS and the NINDB. This had the effect of nationalizing the financing, planning, and control of MS research in the United States and also helped to solidify hierarchies in the specialty of neurology in America. This was not due simply to the increased role of the federal government because it was not clear what was public and what was private because of the *de facto* merging of the voluntary societies with the NINDB. What emerged was a national research conglomerate with multiple sources of financing under the control of neurologists as a specialty. Though patients were an important force in changing the structure of MS research in the United States it was neurologists who were the immediate beneficiaries of this biomedical expansion. Nevertheless, the popular crusade of the NMSS did change the illness experience of many MS patients by ending the patients' isolation by institutionalizing and encouraging collective struggle against the disease.

ENDNOTES

1. Sources include: *Quarterly Cumulative Index Medicus* (Chicago: American Medical Association, 1900-1956). *Index-Catalogue of the Library of the Surgeon-General's Office*, (Washington, D.C.: U.S. Dept. of Health, Education, and Welfare, Public Health Service, 1880-1961). *Current List of Medical Literature, volumes 29-36* (Washington, D.C.: Armed Forces Medical Library, 1956-1959).

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> this computation was given to the Society by me as a guess some years aback when the Association for Research in Nervous and Mental Diseases had their meeting on multiple sclerosis in New York City. The guess was made on the basis of the studies already conducted and Dr. Lindbergh's statistical investigations. At that time I was the chairman of the Statistical Committee for the Society . . . I can assure you that the use of figures in such a manner is really detrimental to the Society. There has also already been an attack upon your figures in an article, which appeared in *Harper's Magazine* in January of this year, dealing with medical statistics. The statement not only discredits the Society but also discredits the National Institute. It is my hope that in future issues of the annual report, or statements to the public, such a statement will not appear again . . . There is no need to plead the cause of multiple sclerosis on the basis of numbers. The longer term disability produced by the illness makes it manifest to me, at least, that it is the most significant neurological problem now existing.

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85. Benge, "I Escaped a Wheelchair," 62.

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135. Letter, Information Resource Center, NMSS, NYC to Colin Talley, San Francisco, CA, 12/31/1997, in possession of author. "$102,000 Medical Gift," 5.

136. "Requests for Admission to the Clinical Center, June 10, 1952 to July 10, 1953," Typescript, 19 pp., NARA/NIH, RG 443, Series 47, Box 14, Folder "1-Patients Admissions, Inquiries (General-(unnamed patients) Filed Date Order."

137. Leonard Scheele, M.D., Surgeon General, USPHS, NIH, National Advisory Neurological Diseases and Blindness Council, Minutes of Meeting, 2/23-24/1952, NARA/NIH, RG 443, Series 12, Box 27, Folder "Committees 1-7 National Advisory Neurological Diseases and Blindness Council Minutes of Meeting 2/23 and 2/24, 1952," 4.

138. Letter, Roswell B. Perkins, Acting Secretary, Dept. Health, Education, Welfare, Washington, D.C. to Congressman Frederick R. Coudert, Jr., 7/5/1955, NARA/NIH, RG 443, Series 47, Box 9, Folder "Hospital I Information (reconstruction, maintenance and operations.)

139. Richard Masland, M.D., Comments, Copy of Minutes of Dec. 8 meeting of "National Committee," 12/8/1960, NARA/NIH, RG 443, Series 48, Box 15, Folder "Organizations and Conferences NMSS, 1956-61".

140. Cannan found that this "new method of supporting research has become

dominant. The bulk of the funds presently available are distributed in the form of grants-in-aid of specifically defined projects which are submitted by individual investigators and evaluated by committees of experts . . . The system of project support is familiar to all of you. It is now deeply entrenched in the contemporary patterns of medical research. All of us who pursue investigations have become intensely project-minded. No one particularly likes the system but no one has yet thought up an acceptable alternative." R. Keith Cannan, M.D., Vice Chairman Division of Medical Sciences, National Research Council, Copy of Speech delivered to American Rheumatism Association, 5/28/53, in Memorandum, Harold R. Wainerdi, M.D., Consulting Medical Director, NMSS, NYC. 6/11/53, to Members of the Medical Advisory Board, NMSS, NARA/NIH, RG 443, Series 47, Box 2, Folder "NMSS, vol. I"

141. USPHS, NIH, National Advisory Neurological Diseases and Blindness Council, Minutes of Meeting, 11/16/50, NARA/NIH, RG 443, Series 12, Box 27, Folder "Committees 1-7 National ADvisory Neurological Diseases and Blindness Council Minutes of Meeting 11/16/1950," 3-4.

142. Letter, Leonard A. Scheele, M.D., Surgeon General, to Harold R. Wainerdi, M.D., Associate Medical Director, NMSS, NYC 2/9/53, RG 443 Series 47, Box 2, Folder "NMSS vol. I."

143. USPHS, NIH, National Advisory Neurological Diseases and Blindness Council, Minutes of Meeting, 11/16/50, NARA/NIH, RG 443, Series 12, Box 27, Folder "Committees 1-7 National Advisory Neurological Diseases and Blindness Council Minutes of Meeting 11/16/1950."

144. Letter, Harold R. Wainerdi, M.D., Medical Director, NMSS, NYC to Pearce Bailey, M.D., NINDB, 4/8/1955, NARA/NIH, RG 443, Series 47, Box 2, Folder "Assoc. I. NMSS, vol II Jan. 54-." USPHS, National Advisory Neurological Diseases and Blindness Council, Minutes of Meeting, June 4, 1951, NARA/NIH, RG 443, Series 12, NIH, National Research Institutes, NINDB, National Advisory Neurological Diseases and Blindness Council Meetings, Nov 50-Nov 54, Box 27, Folder, "Committee 1-7, National Advisory Neurological Diseases and Blindness Council Minutes of Meetings, June 4, 1951." See also, Minutes of Meetings, June 24-25, 1955.

145. Letter, Cornelius Traeger, M.D., NMSS, NYC to Frederick L. Stone, Ph.D., Chief, Extramural Programs, NINDB, Washington, D.C., 11/28/1951, NARA/NIH. RG 443, Series 47, Box 2, Folder, "National Multiple Sclerosis Society, volume I"

146. Letter, Frederick L. Stone, Ph.D., Chief, Extramural Programs, NINDB, Washington, D.C., to Cornelius Traeger, M.D., NMSS, NYC to 12/7/51, RG 443, Series 47, Box 2, Folder, "National Multiple Sclerosis Society, volume I."

147. Letter, Frederick L. Stone, Ph.D., Chief, Extramural Programs, NINDB, Washington, D.C. to Cornelius H. Traeger, M.D., Medical Director, NMSS, NYC 4/10/1952, NARA/NIH, RG 443, Series 47, Box 2, Folder "NMSS, vol. I."

148. Letter, Frederick L. Stone, Ph.D., Chief, Extramural Programs, NINDB, Washington, D.C., to Cornelius Traeger, M.D., NMSS, NYC to 12/3/51, NARA/NIH, RG 443, Series 47, Box 2, Folder, "National Multiple Sclerosis Society, volume I."

149. Letter, Frederick L. Stone, Ph.D., Chief, Extramural Programs, NINDB, Washington, D.C., to Cornelius Traeger, M.D., NMSS, NYC to 12/7/1951, NARA/NIH, RG 443, Series 47, Box 2, Folder, "National Multiple Sclerosis Society, volume I"

150. Letter, Cornelius H. Traeger, M.D., Medical Director, NMSS, NYC to Frederick L. Stone, Ph.D., NINDB, 4/8/1952, NARA/NIH, RG 443, Series 47, Box 2, Folder "NMSS, vol. I"

151. "Heads Medical Unit," *NYT* (8/16/1955): 25:8. Letter, Henry A. Imus, Asst. to the Director, NINDB, to Frederick L. Stone, Ph.D., Scientific and Medical Director,

NMSS, NYC, 9/13/55, NARA/NIH, RG 443, Series 47, Box 6, Folder "6 Morbidity and Mortality (includes reports) alpha by disease)."

152. Letter, Harold R. Wainerdi, M.D., Medical Director, NMSS, NYC to Pearce Bailey, M.D., Director, NINDB, Bethesda, MD, 4/24/54, NARA/NIH, RG 443, Series 47, Box 15 Folder "7-1 Affiliations."

153. Letter, Pearce Bailey, M.D., Director, NINDB to Cornelius H. Traeger, M.D., Medical Director, NMSS, NYC, 11/1/1951, RG 443, Series 47, Box 19, Folder "Research."

154. Letter, Pearce Bailey, M.D., NINDB, to Cornelius Traeger, M.D., NMSS, NYC, 10/2/1952, NARA/NIH, RG 443, Series 43, Box 2, Folder "NMSS vol. I"

155. Letter, Cornelius H. Traeger, M.D., Medical Director, NMSS, NYC to Pearce Bailey, M.D., NINDB, 4/4/1952, NARA/NIH, RG 443, Series 47, Box 2, Folder "NMSS, vol. I." Letter, Pearce Bailey to Cornelius H. Traeger, M.D., 4/8/1952, NARA/NIH, RG 443, Series 47, Box 2, Folder "NMSS, vol. I"

156. Letter, Cornelius H. Traeger, M.D., NMSS, NYC to Pearce Bailey, M.D., NINDB, 4/30/52, NARA/NIH, RG 443 Series 47, Box 2, Folder "NMSS, Vol. I." Letter, Frederick L. Stone, M.D., Medical and Scientific Director, NMSS, NYC to Pearce Bailey, M.D., NINDB, 10/28/55, NARA/NIH, RG 443, Series 47, Box 13, Folder "1-Meetings, Engagements, Invitations, Visits, vol II Jan 53-Dec 54."

157. Letter, Pearce Bailey, M.D., Director, NINDB, to Sylvia Lawry, Executive Secretary, NMSS, NYC, 2/2/1952, RG 443, Series 47, Box 2, Folder, "NMSS, vol. I." Letter, Pearce Bailey, M.D., NINDB, to Roland P. Mackay, M.D., Chicago, IL, 4/2/1952, RG 443, Series 47, Box 2, Folder "NMSS, vol. I." Minutes of the Meeting of the Medical Advisory Board, September 25, 1953, Washington, D.C. Chapter, NMSS in NARA/NIH, RG 443, Series 47, Box 1, Folder "NMSS, Washington D.C. Chapter"

158. Letter, Harold R. Wainerdi, M.D., Associate Medical Director, NMSS, NYC to Pearce Bailey, M.D., Director, NINDB, Washington, D.C., 9/16/1952, NARA/NIH, RG 443, Series 47, Box 2, Folder "NMSS, vol. I."

159. Letter, Ralph C. Glock, President, NMSS, NYC to Pearce Bailey, M.D., Director, NINDB, Bethesda, MD 11/19/1954, NARA/NIH, RG 443, Series 47, Box 2, Folder "Associations 1, NMSS, Vol II Jan 1954 through", 1.

160. "Heads Medical Unit," 8. Letter, Henry A. Imus, Asst. to the Director, NINDB, to Frederick L. Stone, Ph.D., Scientific and Medical Director, NMSS, NYC, 9/13/55, NARA/NIH, RG 443, Series 47, Box 6, Folder "6 Morbidity and Mortality (includes reports) alpha by disease)."

161. Letter, Thomas L. Willmon, M.D., Medical and Research Director, NMSS to Richard L. Masland, M.D., Director, NINDB, 12/23/1959, NARA/NIH, RG 443, Series 48, Box 15, "Organizations and Conferences NMSS, 1956-61."

162. Letter, Thomas L. Willmon, M.D., Medical and Research Director, NMSS to Richard L. Masland, M .D., Director, NINDB, 6/20/1960, NARA/NIH, RG 443, Series 48, Box 15, "Organizations and Conferences NMSS, 1956-61."

163. Letter, Dr. C.H. Traeger, M.D., Medical Director, NMSS, New York, NY to Dr. David Price, NIH, Washington, D.C., 10/26/1948, RG 443, Series 2, Box 142, Folder "0745, Research Grants."

164. Letter, Harold Tager, Jr., Information Officer, NINDB, Washington, D.C. to Howard E. Schaad, Bronx, New York, 12/22/1952, NARA/NIH, RG 443, Series 47, Box 15, Folder "S"

165. Bailey's full quote was as follows:

> early in 1952, representatives of the Institute and of this Society began discussion of the possibility of a pamphlet that would meet both the needs

> of the Society and the responsibilities of the Institute. It was apparent <sic> that two publications should be avoided not only to eliminate waste, but also to avoid the impression that there were differences of opinion between public and private agencies concerned and to avoid almost certain discrepancies in the scientific information which would be made available . . . the decision reached jointly was that the Society rather than the Institute should produce, publish and distribute the pamphlet. There were a number of factors recommending this decision, the primary one being the probably greater acceptance by the medical profession of a private rather than a Governmental issuance and the better facilities of the Society in reaching the patient, the public, and the profession . . . Furthermore, the need at this time appeared to be a pamphlet that would constitute general patient-public orientation, more appropriate for the Society, rather than a scientific report as would be appropriate for the Institute.

Memorandum, Pearce Bailey, M.D., NINDB, to Legal Advisor, NIH, Office of General Counsel, 4/17/1953, NARA/NIH, RG 443, Series 47, Box 11, Folder "1 Decisions, Opinions, Interpretations."

166. Cross-Reference Sheet, Pearce Bailey, M.D., NINDB to Sally Whitcup, NMSS, NYC, 11/22/1955, NARA/NIH, RG 443, Series 47, Box 2, Folder "Associations I NMSS vol II Jan 54-."

167. Letter, Harold R. Wainerdi, M.D., Medical Director, NMSS, NYC to Pearce Bailey, M.D., NINDB, 4/8/1955, NARA/NIH, RG 443, Series 47, Box 2, Folder "Assoc. I. NMSS, vol II Jan. 54-."

168. Letter, Pearce Bailey, M.D., NINDB, Bethesda, MD to Harold R. Wainerdi, M.D., Medical Director, NMSS, NYC, 4/15/1955, NARA/NIH, RG 443, Series 47, Box 2, Folder "Assoc. I. NMSS, vol II Jan. 54-."

169. Letter, Harold R. Wainerdi, M.D., Medical Director, NMSS, to Pearce Bailey, M.D., NINDB, 4/25/1955, NARA/NIH, RG 443, Series 47, Box 2, Folder "Associations I NMSS vol II. Jan 54-."

170. Letter, Pearce Bailey, M.D., NINDB to Harold R. Wainderdi, M.D., Medical Director, NMSS, to 5/10/1955, NARA/NIH, RG 443, Series 47, Box 2, Folder "Associations I NMSS vol II. Jan 54-."

171. Letter, Cornelius H. Traeger, M.D., Medical Director, NMSS, NYC to Members of the Medical Advisory Board-NMSS, 8/29/1952, NARA/NIH, RG 443, Series 47, Box 2, Folder "NMSS, vol. I."

172. Letter, Harold R. Wainerdi, M.D., Medical Director, NMSS, NYC, Members of the Medical Advisory Board, NMSS, 1/12/1954, NARA/NIH, RG 443, Series 47, Box 2, Folder "Associations 1, NMSS, Vol II Jan 1954 through."

173. Ibid.

174. The full quote is as follows:

> The Multiple Sclerosis Foundation of Chicago was organized as an independent foundation, after having consulted us as to the basis for chapter affiliation . . . The program of the M.S. Foundation consists entirely in the financing of a multiple sclerosis research program at Northwestern University for a five-year period at an annual budget of $25,000, under the direction of Dr. Lewis J. Pollock . . . there is no Medical Advisory Board except for personnel of Northwestern University. Therefore, no objective review of this project by a medical body has been effected . . . Our negotiations with the M.S. Foundation, primarily through Mr. Francis

Abeles, its past President and now Honorary President, have been unsuccessful due mainly to our reluctance to concede to their request that they be given equal representation on our Board of Directors . . . In addition, the Chicago Foundation had expressed the desire to spend its funds for research as it sees fit, without submitting its research program to our Medical Advisory Board for approval . . . When Mr. Dunworth spent time in Chicago, he came to the conclusion that the presently constituted members of the Chicago Foundation are not a representative group of people and that the potential resources for M.S. were much higher. He met with Mr. John E. Baker, Jr., partner of the law firm of Baker, Hagstrom & Murray. Mr. Baker's wife has M.S. and his wife's father, a Dr. Theodore S. Proxmire, is a leading physician in Lake Forest Park who has many social contacts. Mr. Baker agreed to organize a M.S. Chapter of our Society under our regulations and expressed a lack of regard for Dr. Lewis Pollock from a personal relationship as well as for his unwillingness to cooperate. Mr. Baker outlined a list of leading social and industrial figures in Chicago who were personal friends of his and among whom he thought he could organize an effective Board of Directors. He did feel that the people active in the M.S. Foundation in Chicago would be suitable additions to his Board (confidential). Mr. Baker, I understand was a classmate of Fred Camp's at Princeton and Alan Breed was also a member of that class. It is Dunworth's opinion that Baker could form an excellent chapter. However, when Baker began to take hold, he was discouraged over the fact that Northwestern, through Dr. Pollock, stated that they would not cooperate with any endeavor other than that of the present M.S. Foundation, of which Francis Abeles was recently succeeded by Mr. Leslie M. Price as President. The attempt to win Northwestern and Pollock over was undertaken by Dr. Hans H. Reese . . . Hans Reese reported that in his opinion Northwestern would become more prone to cooperate if a real powerful chapter were organized in Chicago.

Memorandum, Sylvia Lawry to Oliver E. Buckley, NMSS, 9/8/1952, NARA/NIH, RG 443, Series 47, Box 2, Folder "NMSS vol. I."

175. Lawry's full quote was as follows:

This group recently has expanded its name to give a national implication so that where we have been accustomed to refer to it as 'the Chicago Group' or 'the Multiple Sclerosis Foundation,' it is now designated as the Multiple Sclerosis Foundation of America . . . I have reviewed a very extensive file covering our negotiations with these people. I think that the most important comment that I can make is that they appear to be high-minded and devoted to investigating data about multiple sclerosis, but somewhat unsophisticated in their truculent attitude toward working with a larger group such as ourselves, whose interest is obviously selfless and who have developed extremely intricate controls to insure the quality of work performed under our aegis . . . I would not doubt for a moment the high-minded interests of the lay individuals on the Chicago group. There is an extensive and intricate relationship with Northwestern University about which I believe we still do not have complete details, but the influence of that Institution in the city of Chicago appears to be so dominant that few medical persons in that community were, until recently, willing to work independently . . .We have been approached by a very large number of persons, lay and medical, in

> recent months, who have felt that the type of organization we have, with the Medical Advisory Board and an extensive educational program, best fits the picture . . . It seems to me that there are personality issues involved, some of which are on such a poor level as feeling that they should not surrender hegemony to an Eastern organization . . .

Letter, Harold R. Wainerdi, M.D., Acting Medical Director, NMSS, to Pearce Bailey, M.D., NINDB, 2/16/1953, NARA/NIH, RG 443, Series 47, Box 2, Folder "NMSS vol. I." Bailey had helped to initiate efforts in Chicago in 1952. See Letter, Pearce Bailey, M.D., NINDB, to Roland P. Mackay, M.D., Chicago, IL, 4/2/1952, NARA/NIH RG 443, Series 47, Box 2, folder "NMSS, vol. I."

176. James T. Patterson found the NCI's National Advisory Cancer Council "enjoyed remarkable freedom from political control . . . The scientists . . . implemented the law <creating the NCI> in ways that advanced their own laboratory research and institutional interests . . . ," Patterson, *Dread Disease*, 135-36. Paul Starr found that NIH scientists controlled how they spent their appropriations "to a remarkable degree. The approval of grant applications as well as basic policy issues rested with panels of nongovernmental scientists," Starr, *The Social Transformation of American Medicine*, 343.

177. "Sclerosis Unit Post to Reese," *NYT* (2/29/1952): 27:8.

178. Memorandum, Pearce Bailey, M.D., NINDB to Director of NIH, 10/12/1954, NARA/NIH, RG 443, Series 47, Box 20, Folder "Research 3-2-3 MS Isoniazid Project."

179. Letter, Leonard T. Kurland, M.D., Epidemiologist, Phoenix Mental Health Center, Phoenix, AZ to Dr. Pearce Bailey, M.D., NINDB, 3/6/1952, NARA/NIH, RG 443, Series 47, Box 20, Folder, "Research 7 Epidemiology."

180. Letter, Pearce Bailey, M.D., NINDB, to Leonard T. Kurland, M.D., Epidemiologist, Phoenix Mental Health Center, Phoenix, AZ, 3/17/1952, NARA/NIH, RG 443, Series 47, Box 20, Folder, "Research 7 Epidemiology."

181. Statement of H. Houston Merritt, M.D., NINDB, 1958?, NARA/NIH, RG 443, Series 51, Box 8, Folder "Merritt, Dr. H. Houston." On the question of who ruled American neurology, the answer is that it was that group of neurologists who served in a system of interlocking directorates which controlled the following organizations: the NINDB; American Neurological Association; Association for Advancement in Nervous and Mental Diseases; American Academy of Neurology; American Medical Association, Section on Nervous and Mental Diseases; Veteran's Administration, Section on Neurology; and the medical advisory boards of: NMSS; United Cerebral Palsy; Assoc. for the Aid of Crippled Children; National Society for Crippled Children and Adults, Inc.; National Epilepsy League, Inc.; Muscular Dystrophy Associations of America, Inc.; National Association for Retarded Children; National Society for the Prevention of Blindness; National Neurological Research Foundation. In terms of money outlaid for medical research the primary sources were the voluntary societies and the NINDB.

Chapter Five
The Treatment of Multiple Sclerosis in Los Angeles and the United States, 1947-1960

I A Cultural Approach to Understanding Therapeutics

The study of multiple sclerosis proved to be an excellent probe for understanding the history of clinical practices and therapy in the 1940s and 1950s. Those historians of twentieth-century medicine interested in therapeutics have focused mainly on the rise of the randomized clinical trial and efforts at therapeutic reform. Few historians have analyzed the medical culture of the ordinary practitioner of the early and middle twentieth century.[1]

Harry Marks in *The Progress of Experiment* (1997) described this medical culture mainly as an obstacle to reform which it surely was.[2] However, this medical culture was much more than just an obstacle to reform. It was a fully developed medical cultural system. Medical practitioners in this older medical culture operated under a different set of assumptions and norms than the reformers of the 1920s through the 1950s did. This particular medical culture generated certain styles of therapeutic practice. These therapeutic practices made sense to physicians and patients in the context of their times. In other words, these therapeutic strategies "worked" within the logic of this particular medical cultural system of the 1920s through the 1950s.

Historians using a cultural approach have produced persuasive explanations of nineteenth-century American therapeutics. Representative works have included: Charles Rosenberg's, "The Therapeutic Revolution: Medicine, Meaning, and Social Change in Nineteenth-Century America" (1977); Martin S. Pernick's, *A Calculus of Suffering: Pain, Professionalism, and Anesthesia in Nineteenth-Century America* (1985); John Harley Warner's, *The Therapeutic Perspective: Medical Practice, Knowledge, and Identity in America*, 1820-1885 (1986); and Judith Walzer Leavitt's, "'A Worrying Profession": The Domestic Environment of Medical Practice in Mid-Nineteenth-Century America" (1995).[3]

These works have demonstrated that what constituted healing, how medicine "worked," and the grounds of therapeutic knowledge were historically, culturally, and locally specific; how therapeutic tools were tied to historically specific professional identities; and how physicians' therapeutic decision-making happened through the larger cultural fields of religion, morality, and gender.

One aim of this chapter is to test whether these insights about the culturally bound nature of nineteenth-century therapeutics can be used to interrogate the history of mid-twentieth century therapeutics.[4] My contribution to the history of therapeutics is that I show that a cultural approach provides a useful methodological framework for understanding the history of therapy in the 1940s and 1950s. To what extent can we say that mid-twentieth-century therapeutic practices were culturally and historically specific as well? These questions aim at fleshing out the medical culture of the mid-twentieth century clinic through a synchronic analysis of the field of healing practices. The essential archival source for the data necessary to accomplish this comes from analysis of patient records. In order to understand the treatment of multiple sclerosis in the 1940s and 1950s in the United States I began by analyzing two sets of patient records from Los Angeles County. One group included 227 records from the private practice of Beverly Hills neurologist Tracy Jackson Putnam (TJP) and the other included 86 records from the University of California, Los Angeles Hospital and Neurology Clinic (UCLA).[5]

II Therapeutic Imperative

In America most physicians subscribed to the therapeutic imperative to treat sick patients for reasons of culture, political economy, and professional status. In the United States in response to a diagnosis of chronic disease or as a result of undiagnosed but symptomatic disease patients were active in seeking out physicians through self-referral and in seeking out treatments through self-medication. Even though doctors had no cure for

MS, regular appearances with their physician or neurologist connected patients with the world of science and thus the hope that the culture of the 1940s and 1950s said scientific medicine offered.

Though physicians had garnered unprecedented cultural authority in the 1950s American patients continued to be personally active and assert personal responsibility in dealing with disease. In chronic disease especially many patients' enacted a cultural script wherein it was the sick person's job to be active in overcoming physical and emotional malady through hard work, self-discipline, persistence, positive-thinking, and health activism. Many patients demanded treatment and many offered themselves as human guinea pigs for science. Thus, there was enormous pressure coming from patients which encouraged physicians to engage in therapeutic activism. This was only magnified by a highly decentralized political economy of clinical practice based on fee-for-service billing. The average clinicians' income came from treating individual patients who were clamoring that something must be done.

Most patients shared a cultural preference for therapeutic activism. One patient, a twenty-eight year old photographer from Los Angeles who had had MS for four years, wrote to Tracy Putnam in 1947 reporting that he had written to "Dr. <A>. His answer . . . indicates daily administration of prostigmine by injection followed by one hour of intensive physio-therapy with specialized equipment under the supervision of an especially trained therapist . . . I would like very much to at least give physio-therapy a trial: Obviously I am gaining very little, if any, at present . . . I do not expect miracles, some slight knowledge of the disease, precludes hope of normalcy, but I would like to attain as much as possible."[6]

Patients were more than ready to try therapies regardless of the negative findings in the biomedical literature. A twenty-nine year old man from Orange County wrote Putnam in 1947. The MS patient said, "I visited your office last month and you were going to

write Dr. <name withheld> as to what to give me, I believe it was 'histamine' . . . . As I am quite anxious to do somthing <sic> to try and aleviate <sic> my difficulty I am hoping you can give this your immediate attention"[7]

Some laypersons were even willing to enter the scientific domain of physicians. They did so as individuals and not in an organized way as would happen with AIDs activists in the 1980s.[8] The husband of a fifty-one year old Los Angeles housewife who had had MS for thirteen years sent a research proposal to Putnam in 1952 entitled, "Research Grant for Use of Amino Acids, etc. on Multiple Sclerosis: Case 1: <his wife> . . . Case 2 <his friend> . . . Proposal: Try on patients reducing diets combined with various dosages of nicotinamide various individual amino acids; e.g. Glutamic."[9] This proposal resulted from this layman's observation that "certain elements of Gelatin taken with Vit B and nicotinic acid, etc. are helpful to wife and anxious to see if it will work with other ms pats."[10]

In a May 1954 article in the *Saturday Evening Post*, a thirty-one-year-old multiple sclerosis patient named Robert Grant, Jr. told his story in, "I've Got the Most Mysterious Disease." Grant was a thirty-one-year old veteran of World War II who had been admitted to the VA hospital in Boston in 1948. Grant asked "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."[11]

The husband of a twenty-four year old housewife with MS proposed a surgical procedure for the relief of muscle spasms to a Bellevue Hospital physician. He wrote that the physician's "brother-in-law . . . gave me a copy of the article entitled 'Miracle for a

Nun', concerning the nerve and tendon operation you performed on Sister <name withheld>. Your brother-in-law knew of my interest in Multiple Sclerosis because a mutual friend of ours had told him that my wife has this disease . . . . The thought that occurred to me after reading the article was whether the operation would prevent new muscle spasms occurring as a result of continued inflammations in the central nervous system."[12]

Some patients had a sophisticated knowledge of the latest physiological thinking about MS. One patient wrote Tracy Putnam saying that "enclosed, Dr. Putnam, is a copy of Dr. <A> letter of a few years ago to Dr. <B> of National M.S. It was given to me by a friend who now works for the Society. As you undoubtedly know, Dr. <A> was one of the first researchers to find that demyelinated nerve tissue could be regenerated. That was what he was doing when when <sic> I saw him in his lab at Mt. Sinai Hospital in N.Y. city some three years ago. He did not examine me at the time, but did say he had nothing new then. Since then, however, I understand he has been working on the premise that there well might be in m.s. people an auto-allergy wherein the m.s. patient is attacked from within by his own white corpuscles. Further, that the has been using a leukemia drug, now called '6 MP', and alternately Alkeran. I would like to try it. But only if you think it advisable . . ."[13]

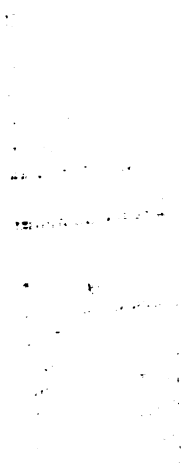
Many American MS patients were very active in searching for physicians who would treat them and in exploring the latest experimental therapies. One MS patient wrote that "if many of us were to make a list of all the medications we had tried we would be defeated by it."[14] A Los Angeles physician described his MS patient's searching for multiple medical opinions as a typical behavior. He wrote that "as frequently happens she began 'working the rounds' and has now come full cycle."[15]

These behaviors and attitudes were not inevitable but generated from American

culture. Part of the popular culture of healing for Americans was to seek out treatments on their own and make pilgrimages to places like Tacoma, Washington where Hinton Jonez gave massive doses of histamine to MS patients in the early 1950s.[16] Because of the operant power of the myth of self-transformation, patients not only sought to heal themselves through vigorous exercises and collective action, many saw it as their responsibility to keep abreast of the latest scientific developments and seek out experimental treatments. Many MS patients did not simply defer to their primary physician as the arbiter of which treatments were valuable but investigated these questions on their own. Doing this often gave the patients more confidence in negotiating treatments with their primary care physicians.

The logic of more activist MS patients who went from physician to physician was similar. MS patient James Rodger remembered a search for a cure in the early 1950s saying that "my wife and I thought it might be wise to try the Tacoma Clinic where, we had heard, much was being tried for M.S. We went to Tacoma with the hope in our hearts that here at last we would find the answer."[17] Just going to the doctor was an act of hope that a cure was around the corner. As one UCLA doctor wrote in the clinical record of a twenty-four year old female patient from Los Angeles in 1960: "Patient comes today wanting to be admitted to the hospital because she wants to get well."[18]

Many patients simply would not accept a nihilistic therapeutic attitude and would insist on treatment.[19] The historian would not see how strong the pressure from patients could be simply by reading the published literature on MS. Close study of patient records proved essential to demonstrating the importance of the patient-physician relationship in explaining therapeutic behavior. Adding weight to the pressure of patients exploiting the decentralized healing system, the cultural artifact of the patient record itself, through a syncretic accretion of narratives of pain and suffering could easily create a moral imperative



to act.[20] For example in this letter from the husband of a thirty-seven year old woman from Anaheim, the husband pleads: "my wife has been declining so rapidly lately that I feel certain that unless you are able to find some means of checking this condition soon it will be too late . . .You are my very last hope but a big hope as I have the utmost confidence in your ability."[21]

Patient records revealed the emotional state of many MS patients which a reading of the popular and medical literature would not show. Most multiple sclerosis patients experienced the first onset of symptoms between twenty and forty years of age. Patient records showed that many young patients attempted suicide or became depressed upon receiving their diagnosis;[22] this created for many physicians a therapeutic imperative to act as can be seen in the following letter from Putnam to a Salt Lake City physician in 1954: "I presume that it is on account of the depression that ACTH and cortisone have not been tried . He is so desperate and despondent, however, that I think a very gradual trial of one or the other might well be undertaken. They could scarcely make matters much worse, and might produce some improvement."[23] The healing culture of patients in the United States created enormous pressure within the medical culture of physicians to treat MS in the 1940s and 1950s.

III Traditional Practices

However, it was not that physicians simply responded to patient pressure and would rather have simply done nothing in the face of MS. Rather, American physicians had a long history of therapeutic activism and the medical culture in the United States encouraged intervention in disease whether a clear physiological cure was known or not. For physicians treating MS in the 1940s and 1950s, and for the patients treated, the specific therapies made sense partly because of the therapies' deep foundations in the traditional

practices of Western ways of healing. The therapeutic practices towards multiple sclerosis from 1946 through 1960 remained powerfully invested with the logic of traditional Western healing. As Guenter Risse has pointed out: "like surviving dinosaurs in a time warp, ancient practices were and and are nevertheless viable and employed."[24] Part of the reason the various vascular pathogenetic theories of multiple sclerosis were persuasive to many in the 1940s and 1950s was that these theories were but the latest packaging for one of the most hoary healing practices in the West: that is, the manipulation of the blood. Whether it was Putnam's blood thinning, or the increased blood flow from vasodilators, or the prevention of blood sludging through Swank's low-fat diet, these strategies made sense, not only in the then current theories of pathogenesis but also because of the powerful roots of these practices in the ancient traditional panoply of Western healing. Putnam and many other physicians advised their clients to move to a warm dry climate to prevent relapses. This was based on what was then considered plausible epidemiological data; that is, there were more cases of MS in northern states than in southern ones; however, because climate was so deeply embedded in the underlying cultural archeology of Western medicine, the recommendation gained added force; and, therefore some patients moved south and west. The same force of cultural tradition applied to therapeutic advice such as dietary manipulations, the proper exercises, hydrotherapy, avoiding fatigue, bed rest, and "morale building." These traditional cultural approaches to treating disease, especially chronic disease, constituted the default-drive of professional therapeutic practices.

This cultural tradition of therapeutic advice also provided foundational support for the particular expression of therapies aimed at hypothetical vascular and auto-allergic pathogenetic mechanisms of demyelination in the 1950s, the two main theories which generated therapies aimed and prevention of relapses. This cultural tradition also provided foundational support for anti-inflammatory (what would have been called antiphlogistic in the nineteenth century) treatments like Adrenocorticotropic hormone (ACTH) during acute

attacks.[25] Further, this cultural tradition supported the use of drugs aimed at symptomatic relief like various sedatives, analgesics, muscle relaxants, antispasmodics, anti-infectives, and laxatives.

These recommendations for treatment from the late nineteenth century provide historical perspective on the treatments proffered during the 1950s. Physician A.B Arnold writing in 1885 stated that "therapeutics has hitherto met with little success in the treatment of sclerosis. If the earlier symptoms of the disease did not usually elude observation, considerable benefit might probably be derived from mild antiphlogistic measures. After the degenerative process is once established there is but faint hope to arrest its fatal tendency. The remedies which deserve some confidence are potassium iodide, nitrate of silver, cod-liver oil and strychnia. Great improvement of the symptoms has been observed from sea bathing and electricity." In 1890, for MS, Arnold recommended "Opium. Among the internal remedies in the treatment of nervous diseases, none are as frequently employed as the narcotics. These substances relieve pain, promote sleep, arrest spasms, and often exert a beneficial influence on the course of many diseases . . . Codeia is a good substitute for morphia, when the latter, owing to idiosyncrasy, cannot be tolerated . . . It must be said in favor of belladonna that it sometimes exerts a sedative effect when morphia fails. Owing to the antagonism of these powerful remedies they are often given in combination . . . useful . . . . in spasmodic affections . . . Hyosciamus. The hypnotic effects of this remedy suggest its use when opium is indicated . . . useful in tremor . . . Canabis Indica sometimes acts well as a hypnotic and in mild forms of neuralgia . . . Chloral hydrate is one of the best hypnotics we possess . . . The bromides form a class of remedies of great value. They lower reflex excitability and exert a general soothing effect on the nervous system. Bromide of potassium alone or in combination with other bromides is our sheet anchor in epilepsy . . . Phosphorus is now much prescribed as a nervine tonic. Its reputation has yet to be established. Strychnia. This powerful excitant of

the axial nerve center was formerly the most favorite remedy in all paralytic affections. Its use has been in great measure superseded by electricity."[26]

Leading neurologist and Professor of Neurology at the New York Post-Graduate Medical School, Charles Dana, wrote in 1892 on the treatment of multiple sclerosis saying that "in the treatment the same measures recommended for other degenerative disease of the nervous system must be employed. Hygienic measures, electricity, and hydrotherapy must be employed . . . Internally the use of large does of iodide of potassium, the hypodermic injection of arsenic, the administration of nitrate of silver and of quinine and other tonics are advised. A very regular, systematic, and quiet mode of life, combined with the use of iodide of potassium and bichloride of mercury, has produced the best results in my experience, even in cases which gave no history of syphilitic infection."[27]

The following therapy examples come from the UCLA Hospital and Neurology Clinic in the 1950s, though they could just as easily come from the 1890s. They demonstrate that physicians deployed traditional practices in the treatment of MS through the 1950s. These treatments had not been proven to be effective through randomized clinical trials but were simply what physicians had "always" done with MS. In 1956 one UCLA physician wrote "Doctor's Order's" in the hospital chart of a thirty-six year old female MS patient indicating that "Pt. may have pre-dinner sherry or bourbon if she wishes."[28] Another neurologist recorded in a patient's examination record in 1955 at UCLA that the "patient has stuck to diet very exactly, and has continued to lose weight in spite of better appetite. He gets tired easily, and cannot walk or work on feet very long. On the whole, however, he says he is better in the way he feels . . . Continue vitamins, diet, and quinine."[29]

Another UCLA physician reported in the "Discharge Summary" of a thirty-four year old female MS patient in 1956 that "the patient was troubled greatly by sleeplessness,

responding very little to the usual hypnotics but fairly well to a mixture of chloral hydrate and whiskey."[30] Another UCLA physician noted in a patient record from 1956 that the "patient continued to improve on the regimen of vitamin C (100mg q.d. and Brewer's yeast (tab XXX q.d.)."[31] A UCLA intern noted in 1956 the patient's "condition on discharge-improvement in spirits."[32] A UCLA Assistant Resident wrote in 1956 that "the patient was treated symptomatically with analgesics and sedatives. Remained afebrile throughout hospitalization and manner gradually became pleasant, tractable and less sarcastic."[33] Another neurologist wrote in 1960 stating that "the patient was urged to take better care of herself, particularly with regard to improving her eating habits relating to breakfast and lunch."[34] The same neurologist prescribed to another patient "Brewer's yeast . . . for general support."[35] Another UCLA physician in 1957 noted in a patient examination record that "in view of evidences of her minor flare up . . . She was cautioned to avoid physical and emotional stresses and strains, rest and continue on low fat diet, Brewer's yeast, and vit C tabs. Ret. 2 mos."[36] Another Assistant Resident in Medicine noted in the "Discharge Summary" of a forty-seven year old housewife from Tuscon, Arizona in 1958 that a senior physician had "suggested that the patient might profit from hydrotherapy and physiotherapy" for pain.[37] A UCLA neurologist wrote in "Doctor's Orders" during the 1958 hospitalization of a forty-year old married male mechanical engineer that the patient should have a "regular diet with supplemental feedings of milk between meals and with cake or cookies."[38]

In the 1940s and 1950s it was the individual clinician who decided which drugs and procedures to try and the individual clinician who evaluated efficacy. The American medical profession valued the autonomous practitioner in a liberal healing system. The unique relationship with the patient gave the autonomous physician legitimate epistemological grounds to evaluate a therapy through a direct if anecdotal encounter in the

clinic. This contradicted with the supposedly universal grounds of modern biomedical knowledge to which therapeutic practices owed pious regard but not necessarily behavioral fealty. This therapeutic activism was supported by a mostly fee-for-service political economy for nonhospital medical expenses in the 1940s and 1950s. Moreover the rapidly growing pharmaceutical industry was quite content with the liberal model of therapeutics because it meant a much more immediate market for its drugs.[39] With this kind of structure, delegitimating anecdotal evidence would be difficult given the enormous cultural power of this model for physicians and patients. The encounter between physician and patient occurred within a cultural system with deep foundations. This culture of healing generated the grammar of the medical approach to the patient and made sense to physicians and MS victims. Therapeutic activism was rooted in a cultural tradition of intervening in disease and was normative for the period.

IV Treatment of MS in 1940s and 1950s

Close study of patient records, physician correspondence, patient correspondence, and published medical literature revealed a continuum of therapeutic activism among physicians with regard to MS in the 1940s and 1950s. At one end of the spectrum were physicians, like Putnam, who attempted to prevent relapses through fairly aggressive treatment using drugs like dicoumarin. At the other end were physicians, like those at UCLA, who attempted to prevent relapses and deterioration through dietary manipulation and lifestyle advice. Most physicians also tried to intervene in the symptomatic complications of MS which might include for example pain, muscular spasms and contractions, and bladder problems. In addition, many physicians in the 1950s attempted to intervene during a flare-up with ACTH. There seems to have been a group of physicians outside of this continuum of therapeutic activism who did not try to intervene in the disease. Popular and medical writers referred to this group as therapeutic "nihilists." It is

not clear how many "nihilists" there were or if the nihilist trope was a straw man because those advocating therapeutic nihilism did not publish in the medical literature. Nevertheless patient letters indicated that there were some physicians who would not treat them; although, this may have been because these physicians were general practitioners and not neurologists and therefore did not feel qualified to treat MS. The sources indicated that once a patient became institutionalized in a place like a Veterans' Administration hospital he or she might only receive custodial care.[40] There were physicians who criticized the panoply of therapies being attempted but they did not go the next step and advocate that nothing should be done to attempt to prevent relapses or empirically address symptoms until better data were available on preventive or symptomatic treatments.[41]

Analysis of two sets of patient records in Los Angeles showed a range of therapeutic activism and clear but different treatment protocols. Putnam's records showed that he used 118 drugs for the treatment of multiple sclerosis while UCLA physicians deployed around 136 drugs. See Tables 10, 11, 12. Putnam and UCLA used 45 drugs in common. See Table 13. Putnam did not use 67% of the drugs which the UCLA physicians prescribed. Likewise, UCLA physicians did not use 62% of the drugs prescribed by Putnam. I grouped the drugs into their properties of physiologic action as described in the pharmacopeias and drug manuals of the period in order to reveal more clearly the different treatment strategies. See Tables 14 and 15. Some drugs may be counted in more than one category. For example, the group listed as sedative also included drugs with hypnotic or tranquilizing effects.

The differences in treatment protocols cannot be explained by differing patient populations because they were roughly similar. See Table 16. For the years 1955 through 1960, the UCLA records showed 88 patients with MS or suspected MS of which I analyzed 86 records. For the same period Putnam saw 67 MS or suspected MS patients of which I analyzed 67 cases. From 1938 to 1972 Putnam saw 227 cases. See Table 17.

Table 10

Drugs TJP Used or Recommended for MS, 1938-72 (most 1947-65), n= 118

ACTH
adenosine
amino acid granules
Ananase
antivert
Apamide
artane sequels
aureomycin
Benzedrine
BetaChlor
calcium gluconate
CCIII
chlortrimeton
chymar
clarin
CO2
codeine
cortisone
cough rx
coumadin
cyclex
cyclospasmol
cysteine
darvon
decadron
Dexedrine sulphate
dexamyl
DialCiba
dicoumarol
dilantin
diuril
donnatol
doxinate
dulcolax
enzar
erythroltetranitrate
evipal
ferrous gluconate
flexin
gantrisin
glycoelixir
heparin
histamine
hydergin
isoniazid
KCl
Kemadrin
Kondremal
liver extract
maalox
Marezine
Marsilid
meprobamate
Metamucil
meratran
metirosten
miradon
Muretran
nardil
nembutal
Neostigmine
niamid
nicotinic acid
noctec
nor-adrenalin
Novocaine
orenzyme
pagitane
panparnit
papaverin
parenzyme
parquasit
parsidol
pennicillin
percodan
phenobarbital
Popkin's medicine
prednisone
Priscoline
probanthine
saluron
Sandalwood Oil
seconal
Senokot
Serpasil
solulexin
somagen
sparine
spartase
steroids
super plenamins
Syncorta
synkamin
Synkavit
testosterone
ThiamineCl
Thigesic
thorazine
Thrombolysin
thyroid
tolserol
tonsetine
trancopal
Trasentine
trichloroethylene
tromexan
urinary antiseptics
valium
varidase
vistaril
vitamins
VitB1
VitB12
VitB
VitB6
VitK
VitE
zinc sulphate

Table 11

Tracy Putnam Drugs Used or Recommended, 1955 through 1960, n=63

ACTH	KCl	Sandalwood Oil
Apamide	Kemadrin	Serpasil
chlortrimeton	Kondremal	solulexin
CO2	liver extract	sparine
cortisone	Marezine	super plenamins
coumadin	Marsilid	Syncorta
decadron	meprobamate	Synkavit
Dexedrine sulphate	Metamucil	ThiamineCl
dexamyl	meratran	thorazine
Dial Ciba	metirosten	trancopal
dicoumarol	miradon	trichloroethylene
diuril	Muretran	tromexan
doxinate	nardil	varidase
dulcolax	nembutal	vistaril
evipal	nicotinic acid	vitamins
ferrous gluconate	orenzyme	VitB1
flexin	parenzyme	VitB12
gantrisin	parsidol	VitB
heparin	prednisone	VitB6
hydergin	probanthine	VitK
Isoniazid	rondremul	VitE

Table 12

UCLA Drugs Used or Recommended 1955 through 1960, n=136

ACTH
alcohol
amphetamine
APC w/codeine
A.P.C. tabs
amphogel
Aristocort
artane sequels
aspirin
atropine
banthine
benadryl
benzoic acid
brewer's yeast
CaCO3
caffeine
calcium gluconate
cascara
castor oil
chloral hydrate
chloromycetin
CO2
codeine
cogentin
compazine
corticosporin eardops
cortisone
darvon
demerol
dexedrine
dexamyl
dilantin
donnatol
doriden
D.O.S.S.
diabinase
dorbane
doxinate
dramamine
dulcolax
empirin
ephedrine sulfate
equinal
Feosol
sodium phosphate
flexin
floraquin
fostex lotion and soap
Furadantin
gantrisin
Gelusil
glucose
glycerin
histamine
hydeltra
hydrocortisone ointment
insulin
isoniazid
K triplex
kaolin
KCl
librium
l glutavite
maalox
magnesium citrate
mandelamine
Marsilid
mephenesin
meprobamate
meratran
methergine
methyl cellulose
midicil
mineral oil
MOM
morphine
NaAmytal
NaBenzoate
NaLuminal
nembutal
Neolin
neomycin
neostigmine
neosynephrine
nicotinic acid
nitroglycerin
Novocaine
nupercainal lozenges
orinase
oxygen
pacatal
paraldehyde
pennicillin
percodan
phenergan
phenobarbital
phenylalanine
phisohex soap
Pitressin
prednisone
Priscoline
probanthine
procaine HCl
pyridium
quinine
Ritalin
Robaxin
scopolamine
seconal
Senokot
Serpasil
soda mint tabs
sodium benzoate
sodium phosphate
SOMA
steroids
stilbesterol
terramycin
tetracycline
thorazine
thyroid
tofranil
TPR
trancopal
trilafon
trilene inhalations
typhoid vaccine
urecholine
urinary antiseptics
vespirix
vitamins
VitB1
VitB12
VitB6
VitC
VitE

Table 13

Drugs Used by TJP and UCLA, n=45

ACTH
artane sequels
calcium gluconate
CO2
codeine
cortisone
darvon
dexamyl
dexedrine
dilantin
donnatol
dulcolax
flexin
gantrisin
histamine
isoniazid
KCl
Marsilid
meprobamate
meratran
MOM
nembutal
neostigmine
nicotinic acid
Novocaine
pennicillin
percodan
phenobarbital
prednisone
Priscoline
probanthine
seconal
Senokot
Serpasil
sodium phosphate
steroids
thorazine
thyroid
trancopal
urinary antiseptics
vitamins
VitB1
VitB12
VitB6
VitE

Table 14

Drugs, Procedures, and Categories of Action

ANTI-COAGULANT

Dicoumarol
Coumadin
Clarin
Heparin
Miradon
Tromexan

ANTI-INFLAMMATORY

ACTH
Chymar
Cortisone
Decadron
Deltra
Meticorten

ANTI-HISTAMINE

antivert
Chlortrimetron
Marezine

DEBRIDEMENT

Parenzyme
Orenzyme
Ananase

VASODILATORS

adenosine
antivert
Cyclospasmol
erythrol tetranitrate
histamine
Hydergin
nicotinic acid
CO_2

ANTI-SPASMODIC

artane sequels
Flexin
Kemadrin
papaverin
Tolserol
Transentine
calcium gluconate

ANTI-CONVULSANT

Dilantin
Nembutal
Noctec
phenobarbital
Valium

MUSCLE RELAXANT

Tolserol
Trancopal
Valium

ANALGESIC

Apamide
codeine
darvon
Novocaine
Percodan
Sandalwood Oil
trichlorethylene
stellectomy
thalamotomy
chordotomy

ANTI-CHOLINERGIC

Pagitane
Panparnit
Parsidol
Priscol
Probanthine
Transentine

NARCOTIC, ANTI-DEPRESSANT, HYPNOTIC, SEDATIVE

codeine
Darvon
Dial Ciba
Evipal
Marsilid
Meratran
Nardil
Nembutal
Niamid
Noctec
Percodan
phenobarbital
Seconal
Serpisil
Thorazine
Valium
Sparine
Tolserol
Trancopal
Vistaril

CNS-STIMULANTS

Benzedrine
Dexadrine sulphate
nor-adrenalin
Neostigmine

DIURETIC/URI-NARY ANTISEPTIC

Diuril
Saluron
Sandalwood Oil

CONSTIPATION

Doxinate
Dulcolax

ANTIBACTERIAL

Aureomycin
Gantrisin
pennicillin

TUBERCULO-STATIC

Isoniazid

ANEMIA

ferrous gluconate
liver extract
vitamins

PROTHROMBINO GENIC

Synkavit
Synkamin
zinc sulphate

LIPOTROPIC

Beta Chlor.

Table 15

TJP and UCLA Drug Classes Compared

	n=86/88 UCLA 55-60	n=67/67 TJP 55-60	n=228/228 TJP 38-72
Sedatives	92%	15%	11%
Analgesics	78%	7%	6%
Laxatives	72%	7%	3%
Antacid/gast.	67%	0	1%
Nutritional	60%	15%	17%
-low-fat diet	28%	0	0
-vitamins	55%	7%	8%
Muscle relax	41%	6%	8%
Antibiotic	40%	3%	4%
Antidepress	26%	7%	6%
Vasodilator	21%	3%	6%
Anti-inflam	19%	63%	40%
Antihistamin	17%	3%	1%
Debridement	1%	19%	11%
Anticoagulan	0	27%	32%

Table 16

Patient Profiles, 1955-1960

SEX	TJP	UCLA
	n=67	n=86/88
F	58%	70%
M	42%	30%

AGE	TJP	UCLA
10-19	0	5%
20-29	18%	16%
30-39	43%	41%
40-49	27%	24%
50-59	7%	13%
60-69	1%	1%
Unknown	3%	0
mean	37 years old	37 years old

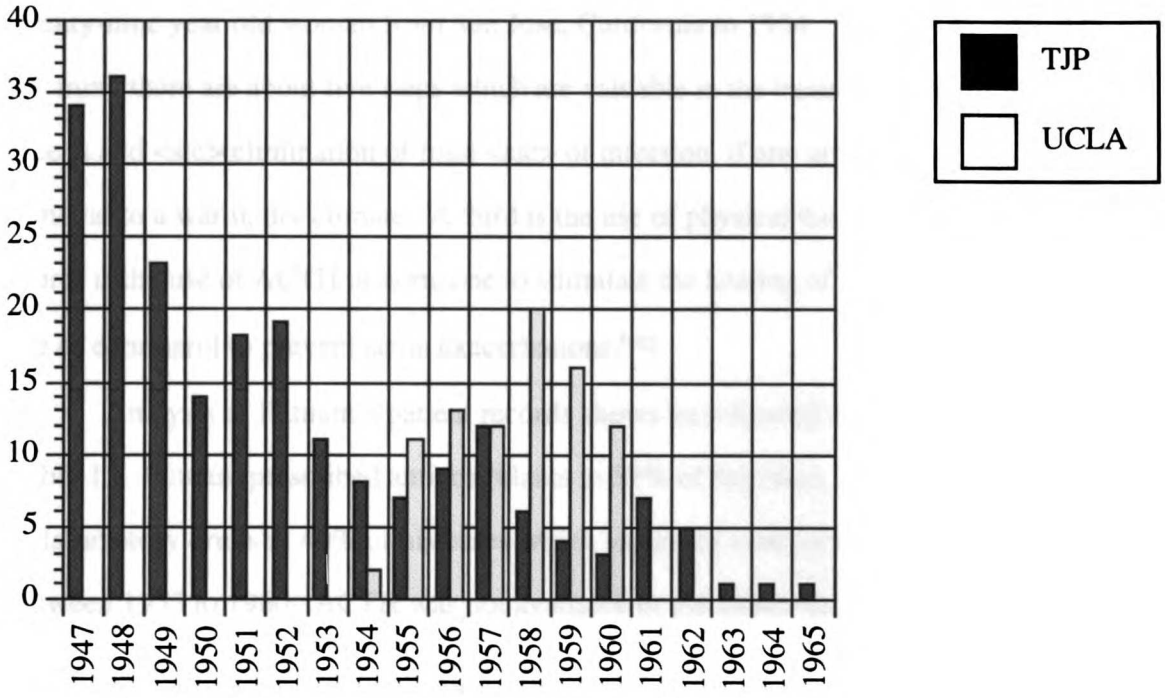
MARITAL	TJP	UCLA
Married	75%	65%
Single	12%	22%
Divorced	3%	6%
Separated	1%	3%
Widowed	3%	2%
Unknown	6%	1%

STATE	n=67	n=86/88
	TJP	UCLA
California	87%	98%
Other	13%	2%

COUNTY	TJP	UCLA
	n=58	n=84
Los Angeles	84%	86%
Southern Cal. not LA	9%	15%
Northern California	5%	0
Unknown	2%	0

Table 17

First Visit by Year, MS Patients



Tracy Putnam held to a consistent protocol for the treatment of multiple sclerosis for the years studied. Putnam described his usual treatment plan in a letter to the husband of a twenty-nine year old woman from San Jose, California in 1954. Putnam wrote that "to my mind there are about five steps which are valuable in the treatment of multiple sclerosis. One is and <sic> elimination of fossi <sic> of infection, if any are present. A second is removal to a warm, dry climate. A third is the use of physical therapy as indicated. A fourth is the use of ACTH or cortisone to stimulate the healing of lesions and a fifth is the use of dicumarol to prevent acute exacerbations."[42]

Analysis of Putnam's patient records shows he followed the advice he gave. See Table 15. Putnam prescribed anticoagulants to 32% of his cases. Putnam deployed anti-inflammatory drugs in 40% of his cases which increased over time to 63% of cases between 1955 to 1960. ACTH was not available in the 1940s. Putnam also prescribed drugs with debridement action in 19% of his cases. Putnam advised vigorous physical therapy to 43% of his patients.[43] See Table 18. Putnam advised 24% of his patients to move to a warmer, drier climate, even from Santa Monica, Pasadena or San Diego to the deserts to the east; and in one case he advised the use of a dehumidifier since moving was impossible.

Table 19 shows more clearly see Putnam's prescribing patterns for the years 1955 through 1960. Putnam divided his cases into three main diagnostic groups: one was chronic/progressive/stationary MS, the second remitting/relapsing MS, and the third suspected MS but not yet diagnosed. All patients were relatively just as likely to receive anti-inflammatory drugs, usually ACTH, especially during an acute episode. However big differences occur with regards to who received anticoagulants usually dicoumarin and sometimes coumadin. Putnam believed that anticoagulants only worked to prevent relapses and was therefore of no use for chronic/stationary/progressive MS patients. The patient records bear this distinction out: 48% of Remitters received anticoagulants while only 13%

Table 18

Comparision of Procedural, Environmental, and Lifestyle Advice

	TJP 38-72 n=228	TJP 55-60 n=67	UCLA 55-60 n= 86
physical therapy	43%	18%	40%
climate	24%	16%	0
elim foci of infection	4%	4%	0
tidal irrigation	3%	2%	2%
enema	1%	3%	15%
avoid fatigue	1%	1%	9%
bed rest	0	0	24%
reassurance	2%	1%	6%

Table 19

TJP Drug Classes, Diagnosis, and Prescribing Patterns 1955-1960

	ALL n=67	Dx=MSProg n=16	Dx=MSremit n=25	Dx=MSother n=21	Dx=Other n=5
anti-inflam	64%	69%	64%	62%	60%
anticoagulan	28%	13%	48%	24%	0
debridement	19%	6%	32%	19%	0
sedatives	15%	13%	16%	16%	0
nutritional	15%	0	32%	5%	20%
-low fat diet	0	0	0	0	0
-vitamins	7%	0	16%	0	20%
analgesics	7%	6%	12%	0	20%
laxatives	7%	6%	12%	0	20%
antidepress	7%	6%	12%	5%	0
muscle relax	6%	0	4%	5%	40%
vasodilator	3%	6%	0	4%	10%
antibiotics	3%	0	4%	0	20%
antihistamin	3%	13%	0	0	0
anatacid/gas	0	0	0	0	0

of Progressives did. The figure for the MS-Other group is 24% which approaches the 28% figure for all combined. Also Remitters were much more likely to receive drugs with a debridement action during an attack which included amino acid granules, orenzyme, parenzyme, or varidase.

Putnam rooted this therapy in his belief about the pathogenesis of MS. He wrote in 1951 that "as you know, ther <sic> is now considerable evidence that a venular thrombosis, probably on an allergic basis, precedes the lesions in the nervous system. It is on this basis that dicoumerol <sic> has been used as a protection against acute relapses, and in my experience, extending over the past eight years, the protection afforded is statistically significant, and the dangers are minimal under proper supervision."[44] In a 1959 Putnam still held to this pathogenic theory writing that " I still feel that this <dicoumarin> is a valuable form of prevention of attacks of multiple sclerosis, and have had good results with it. As in the case of coronary thrombosis, it does not afford complete protection but the likelihood of acute recurrences appears to be decreased."[45] The last time I can document Putnam giving an anticoagulant for MS was 1970 when he was seventy-six years old.[46]

Putnam based his clinical strategy on laboratory work he himself had conducted in the 1930s. In a letter from 1961 he recalled that "in 1929, I transferred my activities from the Department of Surgery to the Department of Neurology <Boston City Hospital and Harvard Medical School> where I was promised a position as neurological surgeon. While waiting for proper surgical facilities to be constructed, I accepted a research assignment in the field of multiple sclerosis, a subject I have pursued ever since. My first work was to read virtually everything which had been written about it, over a thousand books and articles, many of them in foreign languages. I then initiated a series of experiments and observations, and did practically nothing else for the next two years."[47]

From 1929 through 1932, while working under Stanley Cobb at Boston City Hospital, he published several of his experimental studies on the histogenesis of multiple sclerosis which described the experimental production of demyelinated plaques in dogs through the administration of tetanus toxin and coagulants.[48] Putnam continued to publish on this theme from 1934 to 1939 while he was Professor of Neurology at Harvard Medical School and Chief of the Neurological Unit at Boston City Hospital in journals such as *Science*, *Journal of the American Medical Association*, *New England Journal of Medicine*, *Archives of Neurology and Psychiatry*, and the *Annals of Internal Medicine*.[49] From at least 1939 and to 1946, while Director of the Neurological Institute at Columbia University and Professor of Neurology and Neurological Surgery at Columbia University, Putnam experimented with the use of anticoagulants as a preventive treatment for MS.[50] In a 1939 letter from Putnam to a physician from Baltimore, Maryland, Putnam wrote: "I should recommend that he should have as much cysteine hydrochloride by mouth as he will tolerate up to a gram a day . . . The aim of the therapy is, of course, to decrease the tendency of the blood to clot and with careful technique it is possible to demonstrate a delay of coagulation of between 50 and 100% in both instances. 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. I think we cannot expect it to prevent relapses entirely, but I think it makes them definitely less likely to occur. They have been rare in the group we have studied. This, of course, does nothing for the symptoms already in existence, but there is a definite tendency for lesions to heal if they do not extend, and it is reasonable to expect some improvement."[51]

In 1947 Putnam published his study, "Results of Treatment of Multiple Sclerosis with Dicoumarin," in the *Archives of Neurology and Psychiatry*.[52] Beginning in May 1942, Putnam had begun 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, Putnam grouped 27 as cases characterized by the appearance of recurrent, acute, sharply limited attacks and remissions. He defined the second group of 16 cases by their slow downward progression of the disease without well-defined exacerbations or remissions. Putnam treated both groups of patients for at least 6 months and not more than 47 months. He raised the patients' prothrombin time but doctors had to closely monitor it so that hemorrhaging would not occur. The results seemed to indicate that 23 of the 27 acute type cases did not have an acute relapse while the drug was being administered. 9 cases out of the slow progressive group of 16 cases showed no benefit from the treatment.[53] Putnam's conclusion was that dicoumarin prevented relapses if given in sufficient doses. Putnam continued to promulgate this view in the chapter on therapy he wrote for the American Association for Research in Nervous and Mental Diseases' volume on *Multiple Sclerosis and the Demyelinating Diseases.* The original conference was held in December of 1948 and these published proceedings came out in early 1950. The dicoumarin study was the basis for his therapeutic decisions from then on and he never wavered in his belief in the role of venular thrombosis in demyelination from 1930 to 1975 when he died at age eighty-one. From our point of view the study was seriously flawed. It was neither randomized nor double-blind. There was no control group, the sample was tiny, and what passed for statistics were quite peculiar because of the way they tried to account for the spontaneous remission problem. Harry Marks found that "statistical analysis remained the rare exception, not the rule, through the 1940s."[54] When clinical researchers used statistics in the 1940s they often did so in "a ritualistic and uncomprehending way."[55]

The evidence from Putnam's Beverly Hills practice was less than overwhelming for dicoumarin's efficacy in retrospect. He prescribed the drug to 74 MS patients and of these

he followed only 17 closely. Of these 17 patients who Putnam closely followed, based on careful study of the patient records, it can be said that he had only equivocal success. In Putnam's defense the 227 MS cases in his records represented only about 5% of the total patient records in his files. Table 20 shows the average number of MS patients Putnam saw per month in each year from 1947 to 1967. In 1947 he saw less than three MS patients per month on average. Only in 1948 did he see as many as one MS patient per week on average. Between 1947 and 1961 he saw usually somewhere between two and four MS patients per month. It is therefore easy to see how his clinical evidence might not have seemed to him to be have presented contrary evidence with regard the efficacy of his dicoumarin treatment. The number of MS patients was small in the context of the totality of his practice. He did not see enough of them at regular enough intervals for negative evidence to accrue in his mind. Because of this, his belief in the efficacy of the treatment could withstand what to a later analysis seemed clearly equivocal or contradictory evidence.

Nevertheless, Putnam was in the mainstream of neurological practice with regard to his dicoumarin treatment, though on the more activist end of the therapeutic continuum. Other California hospitals and physicians also experimented with dicoumarin in the treatment of MS especially in the late 1940s and early 1950s. Putnam recorded the following in the patient examination record on January 6, 1947: "Pt. was on dicoum- 10d. at hosp. <hospital> (St. Joseph-Orange) <Orange county hospital> & every other d. <day> at ho. <home> for 2 or 3 wks."[56] Writing in 1956, another patient recalled that "the laboratory work was done at the General Hospital <L.A. County> Osteopathic Division about nine years ago. I participated in a dicumerol therapy program for a year or more."[57]

More broadly, how did Putnam's neurological contemporaries in the United States view the study, the dicoumarin therapy, and the vascular pathogenesis theory? In short, many neurologists experimented with Putnam's anticoagulant therapy, at least through the

Table 20

Average Number of MS Patients Putnam Saw Per Month

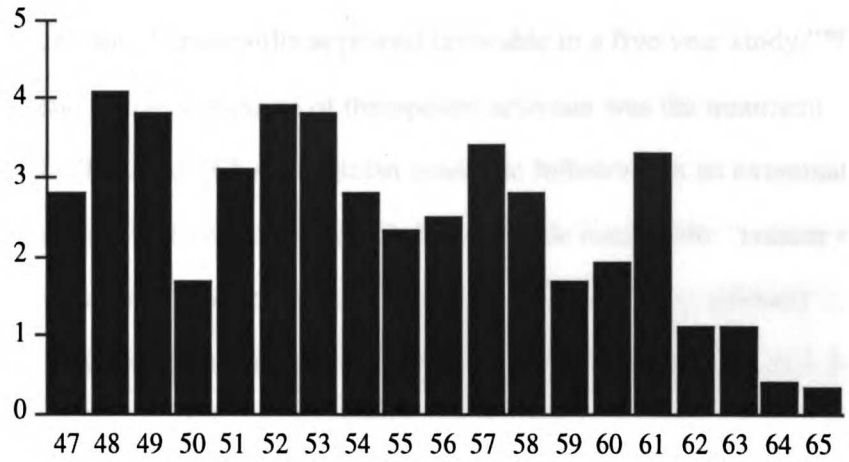


Table 21

UCLA Drug Category and Diagnosis, 1955-60

	n=86 of 88	n=57	n=15	n=9	n=5
TOTAL	ALL	Dx=MS	Dx=Prob.MS	Dx=Poss.MS	Dx=Other
Sedatives	92%	93%	87%	89%	100%
Analgesics	78%	81%	67%	89%	60%
Laxatives	72%	67%	89%	89%	60%
Antacid/gast.	67%	65%	73%	88%	40%
Nutritional	60%	67%	53%	22%	20%
-low fat diet	28%	32%	27%	11%	20%
-vitamins	55%	65%	47%	22%	20%
Muscle relax	41%	49%	20%	22%	40%
Antibiotic	40%	49%	7%	33%	40%
Antidepress	26%	32%	13%	11%	20%
Vasodilator	21%	23%	0	11%	20%
Anti-inflam	19%	25%	7%	0	20%
Antihistamin	17%	16%	13%	22%	40%
Debridement	1%	2%	0	0	0
Anticoagulan	0	0	0	0	0

early 1950s.[58] Richard M. Brickner while Professor of Neurology at Columbia reported in 1948 that "the suggestion of Putnam, that dicoumarin be used for the purpose of avoiding the thrombi he has observed, is being studied on a wide scale. Definitive conclusions have not yet been reached, although the results appeared favorable in a five year study."[59]

On the other end of the continuum of therapeutic activism was the treatment protocol followed at UCLA. A UCLA physician noted the following in an examination record from 1957 of a forty-one year old white Catholic female housewife: "concur with Dr. <name withheld> that Multiple Sclerosis is best bet . . . What pt was advised: . . . that since she might have MS she might as well take the current UCLA Rx regime of 1. low fat diet 2. hi dose Brewers yeast and that if she did have one of the other disorders the same Rx would help. She asked about VitE since she has been a diet fadist <sic>. Was told she could also take this- that it wouldn't do any harm and might help."[60]

Analysis of the UCLA patient records shows that this was the standard protocol at UCLA for MS. These records are from hospital admissions and routine neurological office exams. In terms of preventing relapses UCLA physicians prescribed a high-vitamin, low-fat diet and advised patients to avoid fatigue and often to take bed rest. See Tables 21 and 18. When a patient entered the hospital during an acute attack he or she often received an anti-inflammatory drug like ACTH. Also if the patient was experiencing ocular symptoms UCLA physicians would often prescribe the vasodilator histamine. The UCLA Neurology Clinic gave ongoing primary care to many MS patients, unlike Putnam who functioned mostly as a consulting neurologist. Thus, the UCLA records show heavy dosing of patients with drugs for symptomatic relief. These medications included: sedatives; analgesics; laxatives; antacid medications and drugs for relief of gastric symptoms; muscle relaxants and antispasmodics, for painful contractions, spasms, and stiffness; and antibiotics.

What rationale was there for the preventive approach taken at UCLA, specifically

the high-vitamin, low-fat diet? Neurologist Roy L. Swank of the Department of Neurology and Neurosurgery, McGill University and the Montreal Neurological Institute, supported by a grant from the Multiple Sclerosis Society of Canada, reported on his experience with a low-fat diet and multiple sclerosis in series of articles from 1953 to 1956 in the *Archives of Neurology and Psychiatry* and the *Annals of Internal Medicine* and in one book published in 1961. Swank based his work on a variant of the vascular pathogenesis theory: that is the supposed effects of the sludging of blood incident to fat intake which might lead to demyelination.[61] For example, he wrote in 1953 that "it is further to be noted that high-fat meals decrease the clotting time of the blood. This observation is of interest because of the well-known hypothesis of Putnam that the lesions of multiple sclerosis are due to cerebral venous thrombosis . . . This diet appears to lessen the severity of the disease by reducing the frequency and severity of the exacerbations. Its usefulness seems greatest early in the disease, before significant disability and a steady progression of symptoms have developed . . ."[62] Swank added in 1955 that "the development in animals of increased adhesiveness and aggregation of the red blood cells and of an increased viscosity of the blood after large fat meals . . . will be of importance in the development of this hypothesis."[63] By 1956 Swank had moved to the Division of Neurology at the University of Oregon Medical School in Portland. Becoming more reticent about his diet he wrote that "it can be argued that our reported results are precisely what one would expect from the natural course of the disease: early in the disease patients would remain relatively well, and later they deteriorate. We are unable to disprove this argument, since we do not have a group of patients that we feel can adequately function as controls, nor is such a group of patients available in the literature."[64]

Through 1961 Swank was the only scientist who reported studying the effect of a low-fat diet in multiple sclerosis in the official biomedical literature in the United States.

His data was shaky by his own admission. Nevertheless, the low-fat diet was the standard treatment given to MS patients at the UCLA neurology clinic. This 1959 exchange between two Los Angeles physicians shows that other Los Angeles doctors shared this practice: "I also suggested to him <a thirty-eight year old accountant, married and father of two> that in addition to the nicotinic acid it might be wise for him to be on a polyvitamin preparation. Although the clinical evidence is equivocal, a low fat diet has seemed to be of some help in a few patients with multiple sclerosis and he will be seeing you in a few days in regard to being placed on such a diet."[65]

In addition to the thrombosis theory of Putnam, and the blood-sludging theory of Swank, neurologists tried other treatments based on variants of a hypothesized vascular component to pathogenesis. Richard Brickner, Professor of Neurology at Columbia University, wrote in the *Archives of Neurology and Psychiatry* in 1952 about his new research on vasodilators saying that "many believe that no therapeutic work will be reliable until the fundamental causes of the disease are known. The present study is presented with full recognition of all this, and with all of the caution called for by that recognition . . . . the therapeutic approach suggested . . . is based on a theory of the pathogenesis of the lesions, and this theory is founded on the actual observation of retinal vasospasm associated with scotomas . . . The basic phenomenon of apparent reversibility by vasodilation has been seen at least 134 times, in 34 cases, by four separate observers, at it is thought of as reasonably soundly grounded."[66]

I. Mark Scheinker of the New York Medical College described his therapeutic reasoning in a July 28, 1954 symposium sponsored by the New York Academy of Sciences and the National Multiple Sclerosis Society on the *Status of Multiple Sclerosis*: Scheinker accepted in general the vascular theory of Putnam and added his own observations of low-blood pressure in some MS patients: ". . . the following theory concerning the pathogenesis of multiple sclerosis is suggested: Multiple sclerosis lesions

develop as the result of recurrent episodes of focal disturbance of nutrition by vasoparalytic reactions of the small veins and capillaries of the central nervous system. The vasoparalysis may be preceded by repeated brief periods of vasospasm, as observed by Franklin and Brickner, Grain and Jahsman, Alexander, and others . . . Prolonged and repeated occurrences of vasoparalytic vascular phenomena may result in prolonged stasis and 'sludging' of blood. This effect, in turn, may produce thrombotic occlusion of the small blood vessels and thus, through permanent impairment of the blood supply, give rise to circumscribed patches of demyelination . . . [67] The proposed management of multiple sclerosis is founded upon the writer's theory of the pathogenesis of the disease. These treatments are aimed at elevation of blood pressure and stimulation of the general blood circulation as a counteraction to vasoparalytic vascular phenomena leading to stasis, sludging, and eventual thrombosis of the small blood vessels."[68]

Scheinker then advised the use of vasopressor drugs including: paredrin hydrobromide, desoxyn hydrochloride, and ephedrin sulfate, to be monitored by repeated blood pressure readings; against the vasomotricity of small blood vessels of the central nervous system he suggested: Neo-Calglucon (Sandoz), and Solu-B to increase blood calcium leads in order to increase the tonus of capillaries and arterioles, hoping that this might counteract vasoparalytic vascular distention through the antispasmodic action of calcium. He also recommended Vitamin B complex, testosterone, and buccal Oreton tablets for fatigue. [69]

Treatments based on the vascular theory could have additional rationales as well. One Boston physician in this 1958 letter to a Schenectady, New York physician described his clinical reasoning: the context of which is his acceptance of the possibility of the vascular theory of Putnam or that the disease is a function of enzymatic deficiency: "in view of the fact that this patient has had two other mild attacks but has not fully recovered from

either of his two attacks, I should like to suggest blood transfusion treatment as the treatment of first choice, the rationale being that blood from normal individuals may convey a recovery factor which patients incapable of developing full remissions appear to lack. In those cases the transfused patients show significant improvement compared to matched controls patients."[70]

The vascular hypotheses blended with ideas about a possible allergic factor in MS. This was seen in the multiple uses to which histamine was put, sometimes as a vasoconstrictor and sometimes with reference to the allergic hypothesis. The Mayo Clinic used large doses of histamine on the theory that it would desensitize the MS patients to some auto-allergic reaction implicated as a pathogenetic factor in the formation of sclerotic patches. One Mayo Clinic physician described their treatment this way in 1947: "after completing her neurologic examination, she was sent to my department because of our interest in the experimental study and treatment of this condition. Histamine therapy has been employed in the treatment of multiple sclerosis since June, 1942 . . . During her stay her, she received a total of twenty-seven intravenous histamine injections . . . I did not see <the patient> at the time of her dismissal but Dr. <name withheld>, one of my associates who supervised her treatment during my absence, stated that she made rather remarkable improvement; her paresthesias had decreased to a marked extent . . . I might say that the first five patients to be treated in this manner, have remained essentially well for a period of over four years. That in itself, is interesting, but more time will have to elapse before we can draw any definite conclusions from this form of therapy. Early diagnosis obviously is important because no type of therapy will alter the clinical picture after gliosis has occurred in the central nervous system."[71]

In 1953 the Mayo Clinic continued to give to MS patients "histamine intravenously" according to their "standard method." A Mayo Clinic neurologist described the treatment this way: "she had a total of 27 treatments. We employed a 1:250,000 histamine dilution

'histamine in normal saline'.The rate of adminstration <sic> ranged from 16 to 20 drops per minute. The average amount of histamine which she actually received during a given period of treatment ranged from 40 to 100cc. Treatment was carried out for one and a half hours daily."[72]

Physician Hinton Jonez set up a clinic at St. Joseph's Hospital in Tacoma, Washington specifically to treat MS patients with the same logic employed at the Mayo Clinic. This was done not as a treatment for acute episodes (the way UCLA used it) but as a preventive measure against further attacks. Thousands of patients made the pilgrimage to Tacoma in the late 1940s and early 1950s to receive "the treatment."[73]

Others, perhaps an influential minority, had less sanguine views concerning the treatments discussed so far. Nevertheless, none advocated therapeutic nihilism or a "do nothing" approach. Physician George A. Schumacher, Director of the Neurological Service, Second Cornell Division, Bellevue Hospital and Associate Professor of Clinical Medicine in Neurology and Cornell University Medical College, writing for the Medical Advisory Board of the National Multiple Sclerosis Society in the *Journal of the American Medical Association* in 1950 summarized the state of the art with regards to multiple sclerosis and specifically as regards the use of dicoumarin saying that "many early lesions, consisting only of demyelination and the accompanying phagocytic reaction, are reversible and result in complete recovery of function, thus casting doubt on the role of any specific form of therapy in bringing about the improvement."[74] Specifically on Putnam's dicoumarin treatment study of 1947, Schumacher wrote that ". . . analysis of the figures in the latter study <Putnam's> reveals that the average follow-up time in the series of patients was not as long as the average interval between relapses prior to treatment . . . Severe relapses in the face of adequate dicumarol® therapy and maintained high prothrombin time have been independently reported in 2 patients. The method deserves further study . . ."[75]

Even for drugs used to simply treat the symptoms of MS Schumacher was equally gloomy. He wrote that "the outlook for symptomatic relief by drugs is less optimistic than would appear from the large number of reports which make claims of favorable effects."[76] D. Denny-Brown of the Neurological Unit of Boston City Hospital and the Harvard Neurology Department criticized existing MS treatments, preventive and symptomatic, in 1952: "we have been impressed more by the dangers of giving enough dicoumarin to change blood clotting than by its effect on multiple sclerosis. ACTH and cortisone" use is "in the acute stage of the disease extremely hazardous . . . There is no known potent dietary factor . . . The general use of vitamins and liver extract has no basis in deficiency of factors contained in these. Rather they have replaced arsenic as the stimulator of appetite and well being . . . We find no special reason to recommend B12 and prefer liver extract . . . so far no medicines have been claimed to influence the flexion spasms, dysuria and ataxia, which are the truly difficult problems of the chronic stage of the disease.[77] These criticisms ironically showed the extent to which therapeutic activism was the norm in terms of preventive measures and symptomatic relief.

V Boundary Between Experimentation and Therapy

Indeed, what was an individual clinician to do with a suffering patient in front of him in the clinic? How many would actually send someone home with hopelessness as the only response? Therapeutic activism seemed logical, ethical, and scientific because the distinction between experimentation and therapy during the late 1940s and 1950s was very blurry and what counted as "scientific" in the clinic was different. It was not until the 1960s that the separation of what was experimentation and what was therapy in the clinic became clearer though this began to happen in the United States in some places, like the National Institute for Neurological Diseases and Blindness, in the late 1950s. David Rothman has argued that the roles of researcher and physician and the sites of the

laboratory and clinic were not clearly differentiated in the 1950s. This led to the "confusion of experimentation with therapy."[78] The history of MS in the 1950s corroborates this argument. However, my point is that a particular medical culture valued and supported this conflation of experimentation and therapy. Moreover many patients shared this utilitarian, pragmatic, and experimental view of therapy in the 1950s. So, it cannot be argued in a simplistic way that paternalist physicians experimented on docile patients. Rather, a popular culture of health and American professional medical culture valued therapeutic activism and clinical experimentation.

Most clinicians, like Putnam, grounded their therapeutic decision making in their own biomedical knowledge and in their own clinical experience and considered this "scientific." Because they knew or thought they knew the physiology of the disease, they felt confident in their ability to judge the physiologic effect of drugs in the clinic. Harry Marks found that after World War II medical researchers returned "to research of their own inspiration and under their own control."[79] This medical culture "prized individual experience and judgment above all else."[80] "Clinical investigators thought in terms of the individual scientist, using his intellect to observe and master the phenomena of nature."[81] For this group, as long as one had a legitimate and plausible physiological theory in hand one could experiment with drugs in the clinic. Susan Lederer has demonstrated that there was a tacit ethic which approved of experimentation in the clinic as long as there was hope of therapeutic benefit and no harm would result to the patient before the Second World War. My argument is that this tacit ethic guided clinical behavior through the 1950s as well.[82] The power for determining efficacy lay not in the federal bureaucracy and in statistical methods, as it would later, but with the autonomous practitioner in his own clinic. Nevertheless, during the 1950s there was increasing anxiety and conflict around the grounds of clinical decision-making. The impact this had on restraining therapeutic

activism, at least with regard to MS, was negligible.

The history of MS in the 1940s and 1950s demonstrated this conflation and experimentation and therapy and the way it was valued and supported by ordinary physicians and patients. For example, in a letter from Putnam to a Baltimore, Maryland physician in 1940, Putnam showed approbation toward clinical experimentation: "Dear Dr. <name withheld>: I know of no valid basis for the use of alpha tocopherol in multiple sclerosis, and though some of my friends here are using it in this (and almost every other) condition, I am not impressed with the results. It is apparently harmless, however, and I do not object to it."[83] This comment from a letter from Putnam to a Vista, California physician in 1947 showed approval for clinical experimentation as well: "I, personally, doubt whether the neostigmine has anything to do with the results but certainly there is no harm in trying it. If I can be of any help in outlining further treatment for the patient please let me know."[84]

American physicians mostly accepted this cultural logic with respect to the treatment of MS. Hans H. F. Reese, Professor of Neurology and Psychiatry at the University of Wisconsin Medical School, Madison, wrote in 1949 that while "there is no specific therapy known for multiple sclerosis. Treatment with various vasodilators . . with histamine . . . with dicumarol (Tracy Putnam) are used. In my investigations of multiple sclerosis . . . I have not observed any changes in the coagulability and in the prothrombin time. Nevertheless, we have tried dicumarol for years without any definite benefit."[85] One University of California Hospital, San Francisco neurologist in a letter to an Oakland physician on December 1, 1948 described his choices in the treatment of MS: ". . . Early Multiple Sclerosis. No treatment of any kind offers hope of influencing the process of the disease, or of preventing future attacks. Still, the following therapy may be tried. A course of vitamin B and nicotinic acid, and then arsenic treatment: 20 injections of 200 mgm

sodium cacodylate . . ."[86]

There was no statistical evidence in the literature that any of these remedies were effective and no evidence for the role of an infectious agent which would have been the rationale for the use of neoarsphenamine. Some physicians deployed the antituberculosis drug isoniazid against multiple sclerosis, again, with no evidence of efficacy or an infectious agent's role in MS. The following from a Los Angeles physician to a UCLA neurologist shows the blurry boundary between experimentation and therapy in the 1950s: "the history and neurological findings were compatible with the diagnosis of progressive multiple sclerosis. The mother of the patient was so informed, and she requested that I withhold the information from the patient . . . About one month ago, since working in this clinic, I called her family, an asked whether they might not wish to try some isoniazid, emphasizing that its beneficiary effects were very questionable, but that current investigation considered it worthy of a trial. This apparently set off their desire for help . . ."[87]

This conflation of experimentation and therapy held not only for the more controversial preventive treatments like dicoumarin, histamine, blood transfusions, and low-fat diets but also in therapies directed at the symptoms of MS. One of the vexing problems of handling MS cases was how to deal with painful muscular spasms and contractions. Some physicians deployed quinine for this. In 1954 one Minnesota neurologist recommended that "because of the troublesome extensor spasms of her lower extremities, we would recommend a trial of quinine grains three times a day to see if they would relive <sic> the symptoms. In addition, because the chief disturbances lie in the spinal cord, we would recommend either parenteral liver or B12 therapy. We would also recommend that <the patient> be placed on a low-fat diet . . ." [88]

At the UCLA Neurology Clinic I documented that physicians gave 11 MS patients

quinine from 1955, when the hospital and medical school opened, through 1960. Yet the patient records record little success with this remedy; and there was little or only tepid support for its use in the literature. Richard Brickner commenting in the journal the *Medical Clinics of North America* in 1948 wrote that "the use of quinine, which I suggested some years ago, is probably not very effective."[89] George Schumacher writing about the use of antispasmodics in general in the *Journal of the American Medical Association* in 1954 he declared the following: "antispasmodic Drugs: . . . Quinine is no longer considered useful for this purpose . . . The use of neostigmine would appear to rest on false basic tenets of central action. Favorable reports of its clinical application are poorly controlled . . . The oral use of mephesesin to reduce spasticity, thereby improving muscle power, has not been followed by significantly good results in multiple sclerosis and has in the experience of several observers been complicated by undesirable side effects."[90]

Nevertheless, UCLA physicians still were prescribing quinine in 1960. They did this despite generally poor results. One UCLA neurologist described quinine's effect in 1955 in an outpatient record: "patient took quinine until 2-3 weeks ago. Noted no particular change, except for equivocal increased limberness getting in and out of the bathtub. Otherwise, unchanged."[91] Another UCLA neurologist described quinine's efficacy in a patient record from 1958: the patient "called Dr. <name withheld> and me separately re severe pains in legs, not being relieved by quinine. Have decided to Rx priscoline 25mgs tid empirically."[92] A different UCLA neurologist characterized quinine's use in a patient record in 1958: "no honest objective alteration in neurologic state. The KCl, Quinine, and Artane have done little to improve spasticity."[93] Yet another UCLA neurologist evaluated the results from symptomatic medications in 1960: "banthine did not help her bladder control. Quinine has relaxed her legs practically not at all. Potassium has had no effect. She feels she is continuing to get slowly worse."[94]

This experimental attitude toward symptomatic relief despite shaky or contradictory evidence was widely shared. Putnam recorded this note in patient record from 1955: "talked to Dr. <name withheld> re: his query of the value of hypnotism in these cases. My reaction was unfavorable though admitting I knew of no such work. It was intended to reduce the anxiety- induced aggravation of the tremors. Trial (I believe) would do no harm but (emphasis) trial only!!"[95] This conflation of experimentation and therapy was a strong and persistent cultural formation and was normative for the period in question.[96]

VI Conclusion

In conclusion, for this study of the treatment of MS I have tried to compare and construct the therapeutic behavior patterns in several ways: one has been to document patterns of clinical behavior based on patient records. I then correlated this data with linguistic behaviors such as: letter writing, between physicians and between physicians and patients, journal and book writing, the recording of neurological exams, and with speaking behaviors such as notes on phone calls and what physicians recorded patients said in the clinic. By correlating these meaning filled behavior patterns, in the aggregate, it became clear that all were part of a cultural field of medicine and a popular culture of healing that valued therapeutic activism and clinical experimentation by individual practitioners. The humanitarian narrative of the patient record and strong pressure from patients to treat created a therapeutic imperative which supported the norm of therapeutic activism. The abiding cultural power of traditional therapeutic practices also supported the continuum of behavior therapeutic activism. Given the highly decentralized political economy of the American healing system it is not surprising that one would find multiple and contradictory health practices. The fee-for-service political economy of clinical practice contributed to a medical culture in which the autonomous clinician decided efficacy. These structural conditions made possible varied and contradictory therapeutic approaches to MS in the

ENDNOTES

1. Guenter B. Risse, "The History of Therapeutics," *Clio Medica* 22 (1991): 3-11.

2. Harry Marks, *The Progress of Experiment: Science and Therapeutic Reform in the United States, 1900-1990* (New York: Cambridge University Press, 1997), 46-57, 68, 89.

3. Rosenberg's, "The Therapeutic Revolution: Medicine, Meaning, and Social Change in Nineteenth-Century America" can be found in Charles E. Rosenberg, *Explaining Epidemics and Other Studies in the History of Medicine* (New York: Cambridge University Press, 1992), 9-31. John Harley Warner, *The Therapeutic Perspective: Medical Practice, Knowledge, and Identity in America, 1820-1885* (Cambridge, MA: Harvard University Press, 1986). Martin Pernick, *A Calculus of Suffering: Pain Professionalism, and Anesthesia in Nineteenth-Century America* (New York: Columbia University Press, 1985). Judith Walzer Leavitt, "'A Worrying profession': The Domestic Environment of Medical Practice in Mid-Nineteenth-Century America," *Bulletin History Medicine* 69 (1995): 1-29. For the cultural method applied to other aspects of the history of medicine see Charles Rosenberg and Janet Golden, ed., *Framing Disease: Studies in Cultural History* (New Brunswick, NJ: Rutgers University Press, 1992). Charles E. Rosenberg, *The Care of Strangers: The Rise of America's Hospital System* (Baltimore: Johns Hopkins University Press, 1987). James T. Patterson, *The Dread Disease: Cancer and Modern American Culture* (New York: Harvard University Press, 1987).

4. The late historian of science Thomas Kuhn wrote: "concerned to reconstruct past ideas, historians must approach the generation that held them as the anthropologist approaches an alien culture. They must, that is, be prepared at the start to find that the natives speak a different language and map experience into different categories from those that they themselves bring from home." Thomas S. Kuhn, "Revisiting Planck," *Historical Studies in the Physical Sciences* 14 (1984): 246. Anthropologist Paul Rabinow has emphasized the need "to 'anthropologize the West: show how exotic its constitution of reality has been: emphasize those domains most taken for granted as universal (this includes epistemology and economics): make them seem as historically peculiar as possible: show how their claims to truth are linked to social practices and have hence become effective forces in the social world,'" in Paul Rabinow, "Representations are Social Facts," in J. Clifford and G. Marcus, ed., *Writing Culture: The Poetics and Politics of Ethnography* (Berkeley: University of California Press, 1986), 241, quoted in Paul Rabinow, *Essays on the Anthropology of Reason* (Princeton: Princeton University Press, 1996), x.

5. Tracy Jackson Putnam, M.D. Collection, 1938-1975, Manuscript Collection, no. 90, Special Collections, Louise Darling Biomedical Library, University of California, Los Angeles, CA <hereafter *TJP Collection*> and University of California, Los Angeles Hospital Records <hereafter *UCLA Hospital Records*>. References to patients and physicians are coded. Interested researchers may contact Louise Darling Biomedical Library and the Medical Records Department of the UCLA Hospital for further information. I also analyzed patient records from the New York Hospital for 1919 to 1927 for chapters two and three. Historians of clinical practice in the United States for this period have mostly examined questions of therapeutic reform at the level of discourse rather than clinical practice. See for example, Marc Berg, "Turning a Practice into a Science: Reconceptualizing Postwar Medical Practice," *Social Studies of Science* 25 (1995): 438. Marcia L. Meldrum, "'Simple Methods' and 'Determined Contraceptors': The Statistical Evaluation of Fertility Control, 1957-1968, " *Bulletin History Medicine* 70 (1996): 267-68.

Harry M. Marks uses a structural and organization approach to explain therapeutic reform in, "Notes from the Underground: The Social Organization of Therapeutic Research," in *Grand Rounds: One Hundred Years of Internal Medicine*, ed. Russel Maulitz and Lang (Philadelphia: University of Pennsylvania Press, 1986). For the importance of patient records in medical history see: John Harley Warner, Review of *The Private Science of Louis Pasteur*, by Gerald L. Geison *Bulletin History Medicine* 70 (1996): 718. Joel D. Howell, *Technology in the Hospital: Transforming Patient Care in the Early Twentieth Century* (Baltimore: Johns Hopkins University Press, 1995), 19-20. Guenter B. Risse and John Harley Warner, "Reconstructing clinical activities: Patient records in medical history," *Social History of Medicine* 5 (1992): 183-205. Guenter B. Risse, "The History of Therapeutics," *Clio Medica* 22 (1991): 3-11.

6. Letter, P207, Los Angeles <hereafter LA> to Tracy Jackson Putnam <hereafter TJP>, Beverly Hills,CA <hereafter BH> 12/4/47, Box 23, Folder P207.

7. Letter, P127, Santa Ana, CA to TJP, BH, 5/8/47,*TJP Collection*, Box 14, Folder P127.

8. Steven Epstein, *Impure Science: AIDs, Activism, and the Politics of Knowledge* (Berkeley: University of California Press, 1996).

9. Letter, P179's husband, Pasadena, CA to TJP, BH, 8/22/52, *TJP Collection*, Box 21, Folder P179.

10. Unsigned note, on TJP stationary, presumably August 1952, *TJP Collection*, Box 21, Folder P179.

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

12. Letter, P80's husband, BH, CA to #8, M.D., Bellevue Hospital, NYC, 10/16/63, *TJP Collection*, Box 10, Folder P80. The Bellevue physician responded saying that "it is difficult to outline the principle of the surgical procedure without going into great technical detail. I believe that the 'Gamma system' plays a more important part in neuro-muscular action than we know. The operation is based on cutting down the afferent supply and this results in a secondary change in the efferent system. At this point it is not a procedure for pure placidity- it has been done in tension ataxia, rigidity as well as the spasticity. We have noticed complete muscle realignments following the most simple procedures. We have had no miracle cures. We have, however, made living more bearable and added to the dignity of the patient . . . I hope that we will see the day when a tablet will do the trick. Certainly there was a time when myasthenia gravis was felt to be as hopeless as M.S," Letter, #8, M.D., Bellevue Hospital, NYC to P80's husband, BH, CA, 10/31/63, *TJP Collection*, Box 10, Folder P80.

13. Letter, P60, Utica, NY to TJP, BH, 7/19/68, 1 of 2, *TJP Collection*, Box 8, Folder P60.

14. James Rodger, *The Silent One: The Autobiography of James Rodger* (North Dakota Chapter, National Multiple Sclerosis Society and St. Francis Home, 1965), 39.

15. Letter, #11, M.D., LA, CA to TJP, BH, 2/10/58, *TJP Collection*, Box 13, Folder P112.

16. Hinton D. Jonez, *My Fight to Conquer Multiple Sclerosis* (New York Messner, 1952). Rodger, *The Silent One*, 37-41.

17. Rodger, *The Silent One*, 37.

18. #116, M.D., Physician's Notes, 9/01/60, *UCLA Hospital Records*, Folder U257.

19. Putnam's patient files contain large numbers of letters from patients who read about Putnam in the popular press and requested an appointment with him. See for example, Letter, P29, Long Beach, CA to TJP, BH 6/3/1947, *TJP Collection*, Box 4,

Folder P29. Letter, Mrs. P207, LA to TJP, BH, 2/27/1948, *TJP Collection*, Box 23, Folder P207. Letter, P5, Long Beach, CA to TJP, BH, 3/22/1948, *TJP Collection*, Box 2, Folder P5. Letter, P109, Santa Monica, CA, to TJP, BH, 5/10/1948, *TJP Collection*, Box 12, Folder P109. Letter, P35, Fresno, CA to TJP, BH, 12/26/1950, *TJP Collection*, Box 6, Folder P35. Letter, P4, Westminster, CA to TJP, BH, CA, 2/25/1948, *TJP Collection*, Box 2, Folder P4.

20. Thomas W. Laqueur, "Bodies, Details, and Humanitarian Narrative," in *The New Cultural History*, ed. Lynn Hunt (Berkeley: University of California Press, 1989), 176-204.

21. Letter, P226's husband, Anaheim, CA to TJP, BH, 1/29/47, Box 26, Folder P226.

22. See Patient Examination Record <hereafter PER>, 8/26/1952, *TJP Collection*, Box 5, Folder P31. PER, 5/29/1958, *TJP Collection*, Box 9, Folder P65. PER, 9/4/1947, *TJP Collection*, Box 10, Folder P75. PER, 7/17/1962, *TJP Collection* , Box 10, Folder P80. PER, 5/11/1949, *TJP Collection* , Box 10, Folder P82. TJP, "Hospital Report," 12/15/1961, *TJP Collection* , Box 12, Folder P107. Letter, #27, M.D. Beverly Hills, CA to Dr. #28, LA, CA 5/21/48, in *TJP Collection*, Box 5, Folder P34. Letter, TJP to #19, M.D., San Francisco, CA , 1/24/56, *TJP Collection* , Box 19, Folder P158. Letter, TJP to #17, M.D., Salt Lake City, UT, 1/8/54, *TJP Collection* , Box 19, Folder P162. See also "Physician's Notes," 06/18/1963, Folder U246, *UCLA Hospital Records*."Intern's Admission Note," 3/18/56, Folder U 235,*UCLA Hospital Records*. "Physician's Notes," 4/5/1956, Folder U255,*UCLA Hospital Records*. "Physician's Notes," 3/27/62, Folder U213,*UCLA Hospital Records*.

23. Letter, TJP, BH to #17, M.D., Salt Lake City, UT, 1/8/54, *TJP Collection*, Box 19, Folder P162.

24. Risse, "The History of Therapeutics," 3.

25. Writing in 1995, physician Richard Lechtenberg pointed out that "it is generally accepted that the long-term outcome after the early use of corticosteriods is similar to what would have happened if they had not been used at all. Whether or not there are short-term advantages to using corticosteroids, aside from the obvious ones of reducing discomfort and shortening the duration of flare-ups has been more difficult to resolve," Richard Lechtenberg, *Multiple Sclerosis Fact Book* (Philadelphia: F.A. Davis Company, 1995), 85.

26. A.B. Arnold, *Manual of Nervous Diseases* (San Francisco: The Bancroft Company, 1890, revised second edition), 50-53.

27. Charles L. Dana, *Text-Book of Nervous Diseases Being a Compendium for the Use of Students and Practitioners of Medicine* (New York: William Wood & Company, 1892), 379.

28. #108, M.D., "Doctor's Orders,"3/19/56, *UCLA Hospital Records*, Folder U235.

29. #115, M.D., PER, 2/16/55, *UCLA Hospital Records*, Folder U244.

30. #106, M.D., "Discharge Summary," 4/10/1956, *UCLA Hospital Records*, Folder U255.

31. #103, M.D., Physician's Notes, 6/14/1956, *UCLA Hospital Records*, Folder U303.

32. #110, M.D., Intern, Physician's Notes, 3/22/1956, *UCLA Hospital Records*, Folder U235.

33. #114, M.D., Asst. Resident, UCLA, "Discharge Summary," 6/25/1956, *UCLA Hospital Records*, Folder U235.

34. Letter, #107, Jr. M.D., UCLA to #112, M.D., Hemet, CA, 7/13/60, 3, *UCLA*

Hospital Records, Folder U237.

35. #107, M.D., Physician's Notes, 5/15/1958, *UCLA Hospital Records*, Folder U242.

36. #103, M.D., Physician's Notes, UCLA Neurology Clinic, 1/31/1957, *UCLA Hospital Records*, Folder U243.

37. #105, M.D, Asst Res. in Med., "Discharge Summary," 6/29/1958, *UCLA Hospital Records*, Folder U245.

38. #115, M.D., "Doctor's Orders", 9/18/1958, *UCLA Hospital Records*, Folder U305.

39. Paul Starr, *The Social Transformation of American Medicine: The Rise of a Sovereign Profession and the Making of a Vast Industry* (New York: Basic Books, 1982), 290-378.

40. "<patient>is now at a Veteran's Hospital in New York City. Whether<sic> his condition has deteriorated considerably since you saw him. Although he is not fully a bed patient, he is physically incapacitated, his vision is becoming impaired, as well as his speech. Furthermore, his psychological state and motivation at this point is pitiful-especially since he feels that nothing is being done for him at the Hospital. We are at our wits ends. Knowing <patient's> initial condition, I am wondering what course of action you would advise for P31 at this time . . . We are indeed in a predicament with <patient>. 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?" Letter, P31's brother, Berkeley, CA to TJP, BH, 6/29/55,*TJP Collection*, Box 5, Folder P31.

41. Even George Schumacher, who was the published neurologist most critical of therapeutic practices in the 1940s and 1950s was not completely opposed to empirical experimentation in the clinic. From the University of Vermont in Burlington in 1952 he wrote that "for the practitioner who wishes to leave no stone unturned in his therapeutic approach to the present patient with multiple sclerosis and who is desirous of applying any potentially valuable but unsubstantiated specific method of therapy, reference is made to previous reviews of treatment one of which is also distributed in manual form by the National Multiple Sclerosis Society," George A. Schumacher, "Forward: Symposium on Multiple Sclerosis and Demyelinating Diseases," *American Journal of Medicine* 12 (1952): 500.

42. Letter, TJP, BH to P54's husband, San Jose, CA 8/5/1954, *TJP Collection*, Box 7, Folder P54. This was Putnam's standard advice. Putnam advised that "if this is the beginning of a new attack, I think we might try to head it off by giving her dicoumarol. I presume you are familiar with the technique. We usually use it in doses to bring the prothrombin level down to about 20% and maintain it that way indefinitely. This seems to diminish greatly the tendency to fresh outbreaks but of course does nothing for established symptoms . . . ," Letter, TJP, BH to Dr. #9, Kenmare, ND, 8/4/1949, *TJP Collection*, Box 4, Folder P19. In 1951 Putnam wrote: "I was also wondering if you would take this patient in hand and prophylactically dicoumoralize her within therapeutic levels in an attempt to prevent any further exacerbations," Letter, TJP, to Dr. #12, Fresno, CA, 9/12/51, *TJP Collection*, Box 5, Folder P35. In 1953 Putnam wrote that "my usual advice in slowly progressive cases of multiple sclerosis is to try a course of ACTH and perhaps cortisone, to attempt some intensive physical therapy, and if possible perhaps most important of all, to move permanently to a warm dry climate," Letter, TJP, BH to Dr. #14, BH, CA, 9/30/1953,1, *TJP Collection*, Box 8, Folder P62. In 1958 Putnam wrote that "the reason I feel it is so important to make a definite diagnosis in multiple sclerosis is that we now have available a number of forms of treatment, each helpful in some cases. The most generally effective one is removal permanently to a warm, dry climate, preferably one of those where multiple sclerosis is known to be rare . . . Cortisone seems to me to do as

well as the new substitutes and is a little less expensive. It is my impression, though I am not certain, that the results are better with cortisone that with the newer preparations. Medicine of this general type naturally attack the disorder in its allergic phase. The first recognizable evidence of the appearance of lesions within the central nervous system is the development of thrombi in small veins and these presumable have something to do with the chylomicron index. Some encouraging results have been obtained by the use of anticoagulants, which of course, have no effect on existing symptoms but decrease the tendency to relapses, the statistical benefit being rather better than those cited for the use of dicumerol in coronary disease," Letter, TJP, BH to Dr. #4, Pittsburgh, PA, 1/3/58, 2, *TJP Collection*, Box 10, Folder P84. In 1959 Putnam wrote that "it seems to me that the diagnosis of multiple sclerosis is unquestionable in this case. And the problem is, of course, what should be done for her. I judge that she is unemployable at present and is dependent upon her father. Since she is not tied down to any particular locality, it might be worthwhile to see if a sojourn in a completely arid environment such as Twenty Nine Palms or Palmdale might be helpful to her. While considering this possibility, I believe it would be advantageous to give her a course of ACTH. I usually give 40 units of Acthar Gel daily for ten days, spread over two weeks. If the patient has obtained any improvement form the treatment, I usually follow this with some form of Cortisone. Lately I have been using Decadron in doses of .5 mg. daily. I have had excellent statistical results with the use of Dicumarol or similar drugs in preventing attacks. Her father tells me that the attack of the summer of 1953, occurred while she was taking Dicumarol and he is reluctant to start again. It might be worthwhile to get the old records and see if enough was given to keep the prothrombin percentage around 30." Letter, TJP, BH to Dr. #5, BH, 2/10/1959, *TJP Collection*, Box 12, Folder P102.

43. Putnam said that he was "well aware, also, that many doctors feel that exercise is harmful in multiple sclerosis, indeed this is the old teaching. It is only recently that Dr. Kabat and others have shown that vigorous exercise, under proper supervision, may be helpful," Letter, TJP, BH to P4, Westminister, CA, 3/3/1948, *TJP Collection*, Box 2, Folder P4.

44. Letter, TJP, BH to #6, M.D., Redlands, CA, 1/18/51, *TJP Collection,* Box 26, Folder P222.

45. Letter, TJP, BH to #15, M.D., LA, CA, 3/4/1959, *TJP Collection*, Box 12, Folder P102.

46. Putnam prescribed the anticoagulant coumadin to P161 on 1/13/70. See PER, for P161 on 1/13/1970, TJP, *TJP Collection*, Box 19, Folder 161.

47. Letter, TJP, BH to Mr. Meyer Friedman, BH, 5/16/1961, 2, Box 1, Folder "Biographical Narratives."

48. Tracy J. Putnam, J.B. McKenna, and L.R. Morrison, "Studies in Multiple Sclerosis, I, the Histogenesis of Experimental Sclerotic Plaques and their Relation to Multiple Sclerosis," *Journal American Medical Association* 97 (1931): 1591-95. Tracy J. Putnam, L.R. Morrison , and J.B. McKenna, "Experimental Demyelination," *Transactions of the American Neurological Association* (1931): 451-55. Tracy J. Putnam, J.B. McKenna, and J. Evans, "Experimental Multiple Sclerosis in Dogs from Injection of Tetanus Toxin," *Journal fur Psychologie und Neurologie* 44 (1932): 460-67. Tracy J. Putnam, "The Pathogenesis of Multiple Sclerosis: a Possible Vascular Factor," *New England Journal Medicine* 209 (1933): 89. See also the biographical sketches in *TJP Collection*, Box 1, Folder "Biographical Materials."

49. Tracy J. Putnam, "The Biological Significance of the Lesions of Multiple Sclerosis," *Science* 80 (1934): 295-296. Tracy J. Putnam, "Studies in Multiple Sclerosis, IV, 'Encephalitis' and Sclerotic Plaques Produced by Venular Obstruction," *Archives Neurology & Psychiatry* 33 (1935): 929-940. Tracy J. Putnam, "Studies in Multiple

Sclerosis, VIII, Etiologic Factors in Multiple Sclerosis," *Annals Internal Medicine* 9 (1936): 854-86. Tracy J. Putnam, "Studies in Multiple Sclerosis, VII, Similarities between some Forms of 'Encephalomyelitis' and Multiple Sclerosis," *Archives Neurology & Psychiatry* 35 (1936): 1289-1308. Tracy J. Putnam, "Venous Thrombosis as the Primary Alteration in the Lesions of 'Encephalomyelitis' and Multiple Sclerosis," *New England Journal Medicine* 216 (1937): 103-4. Tracy J. Putnam, "Lesions of 'Encephalomyelitis' and Multiple Sclerosis. Venous Thrombosis as the Primary Alteration," *Journal American Medical Association* 108 (1937): 1477-80. Tracy J. Putnam, "Evidences of Vascular Occlusion in Multiple Sclerosis and "Encephalomyelitis'," *Archives Neurology & Psychiatry* 37 (1937): 1298-1321. Tracy J. Putnam and A. Adler "Vascular Architecture of the Lesions of Multiple Sclerosis," *Archives Neurology & Psychiatry* 38 (1937): 1-15.

50. "It is seldom possible to devise a successful treatment without a knowledge of the fundamental mechanism of the disease. For this reason the first eight years of the work carried out under the Harvard Multiple Sclerosis Fund consisted of a systematic and critical review of the theories of pathogenesis of the disease and an attempt to compare the development of its lesions with pathologic processes of known origin. When the hypothesis of and origin in a thrombosis of small vessels in the nervous system began to intrude itself on the unwilling minds of the investigators, an elaborate series of studies was planned to test the theory from many angles of approach. These included the production of lesions in animals by experimental venous obstruction and by intravenous injection of coagulants, a search for evidences of vascular obstruction in plaques and a comparison of plaques with other pathologic processes . . . . all these investigations confirmed the original hypothesis." During 1939 he experimented with cysteine, heparin, germanin, and certain dyes to test their anticoagulant properties and prophylactic effects. Tracy J. Putnam, "The Criteria of Effective Treatment in Multiple Sclerosis," *Journal American Medical Association* 112 (June 1939): 2491.

51. Letter, TJP, NYC to Dr. #7, Baltimore, MD, 5/13/38, *TJP Collection*, Box 26, Folder P225.

52. Tracy J. Putnam, L.V. Chiavacci, H. Hoff, and H. G. Weitzen, "Results of Treatment of Multiple Sclerosis with Dicoumarin," *Archives Neurology & Psychiatry* 57 (1947): 1-13. Tracy J. Putnam, "The Criteria of Effective Treatment in Multiple Sclerosis," 2488-2491. "Multiple Sclerosis," *Modern Medical Therapy in General Practice* (1940): 2026-2027. "Multiple Sclerosis and 'Encephalomyelitis,"*Bulletin of the New York Academy of Medicine* 19 (1943): 310-16. See also Letter, TJP, BH to Mr. Meyer Friedman, BH, 5/16/1961, 4, *TJP Collection*, Box 1, Folder, "Biographical Materials."

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

54. Marks, *Progress of Experiment*, 136.

55. Ibid., 154.

56. TJP, PER, 01/06/47, 2, *TJP Collection*, Box 26, Folder P226.

57. Letter, P206, Lancaster, CA to TJP, BH, 2/17/1956, *TJP Collection*, Box 23, Folder P206.

58. Hans H. Reese, "Trends in Etiologic Researches of Multiple Sclerosis," *American Journal of Medicine* 12 (1952): 572-73.

59. Richard M. Brickner, "Multiple Sclerosis," *Medical Clinics North America* 32 (1948): 743.

60. #111, M.D., "Physician's Notes," 2/12/1957,*UCLA Hospital Records*, Folder U248.

61. Roy L. Swank, ""Treatment with Low-fat Diet," *Archives Neurology & Psychiatry* 69 (1953): 100-1. Swank's low-fat regime is specifically mentioned in a patient

examination record from 1956 by a UCLA physician: #111, M.D., Physician's Notes, 1/11/1956, *UCLA Hospital Records*, Folder U278.

62. Swank, "Treatment with a Low-fat Diet," 102.

63. Roy L. Swank, "Treatment with low-fat diet: results of 5 1/2 years experience," *Archives Neurology & Psychiatry* 73 (1955): 631.

64. Roy L. Swank, "Treatment with low-fat diet: result of 7 years experience," *Annals Internal Medicine* 45 (1956): 822. See also Roy Swank and Mary-Helen Pullen, *The Multiple Sclerosis Diet Book: a Low-Fat Diet for the Treatment of M.S., Heart Disease, and Stroke* (Garden City, N.Y.: Doubleday, 1977). Swank continued to publish on the low-fat diet until 1987. See Roy Swank and Barbara Brewer Dugan, *The Multiple Sclerosis Diet Book: a Low-Fat Diet for the Treatment of M.S.* (Garden City, N.Y.: Doubleday, 1987).

65. Letter, #117, M.D., LA to #101, M.D., LA, 3/2/1959, *UCLA Hospital Records*, Folder U259.

66. Richard M. Brickner, "Management of acute episodes (with special reference to vasodilating drugs)," *Archives Neurology & Psychiatry* 68 (1952): 197-98.

67. Scheinker, "Circulatory Disturbances and Management of Multiple Sclerosis," 586-88 in "The Status of Multiple Sclerosis," *Annals of the New York Academy of Sciences*, volume 58, art. 5, pp. 541-720, ed. Roy Waldo Miner, July 2, 1954.

68. Ibid., 586-88.

69. Ibid., 588-9.

70. Letter, #2, M.D., Boston, MA to #13, MD, Schenectady, NY, 5/2/58, 3, *TJP Collection*, Box 7, Folder P47. For the approach in general see, Leo Alexander, *Multiple Sclerosis Prognosis and Treatment* (Springfield, IL: Charles C. Thomas, 1961), 74-5.

71. Letter, Dr. #29, Mayo Clinic, Rochester, MN to Dr. #30, LA, CA 1/14/47, 1, *TJP Collection*, Box 9, Folder P68.

72. Letter, #29, M.D., Mayo Clinic, Rochester, MN to #31, M.D., Pasadena, CA 12/24/1954, TJP Collection, Box 12, Folder P102.

73. Jonez, *My Fight to Conquer Multiple Sclerosis* . Rodger, *The Silent One*, 37-41. PER, 11/06/1950, *TJP Collection*, Box 4, Folder P26. Letter, P122's daughter, North Hollywood, CA to TJP, 12/4/1950, 2-3, *TJP Collection*, Box 15, Folder P122. Letter, #32, M.D., San Diego, CA to #33, M.D., San Diego, CA, 10/27/1951, *TJP Collection*, Box 11, Folder P92.

74. George A. Schumacher, "M.S.,"*Journal American Medical Association*143 (1950): 1151.

75. Ibid., 1152. The only other study published in the United States on the use of dicoumarin in multiple sclerosis reported negative results. See H. Kammer, "Mechanism of demyelinating diseases; therapeutic approach with anticoagulants (dicumarol)," *Cleveland Clinical Quarterly* 14 (1947): 153-58.

76. Ibid., 1153.

77. D. Denny-Brown, "Symposium on Multiple Sclerosis and Demyelinating Diseases," *American Journal of Medicine* 12 (1952): 507-8.

78. David J. Rothman, *Strangers at the Bedside: A History of How Law and Bioethics Transformed Medical Decision Making* (New York: Basic Books, 1991), 66.

79. Marks, *The Progress of Experiment*, 111.

80. Ibid., 53.

81. Ibid., 51.

82. Susan Lederer, *Subjected To Science: Human Experimentation in America before the Second World War* (Baltimore: Johns Hopkins University Press, 1995), 9.

83. Letter, TJP, Neurological Institute, NYC, to Dr. #7, Baltimore, MD,

5/24/1940, *TJP Collection*, Box 26, Folder P225.

84. Letter, TJP, BH to Dr. #18, Vista, CA, 8/14/47, *TJP Collection*, Box 7, Folder P51.

85. Hans H. F. Reese, "Diagnosis and Treatment of Multiple Sclerosis," *Postgraduate Medicine* 6 (1949): 130.

86. Letter, #19, M.D., University of California Hospital, San Francisco, CA to #34, M.D., Oakland, CA 12/1/1948, 1, *TJP Collection*, Box 12, Folder P104. See also, Hospital Discharge Summary for P80, Dept. of Neurology, University of California Medical Center, San Francisco, CA, 6/11/1956, 3 in *TJP Collection*, Box 10, Folder P80. Letter, #1 M.D., San Francisco, CA to Dr. #8, Travis Air Force Base, CA, 10/3/1956, in *TJP Collection*, Box 10, Folder P80.

87. Letter, #109, M.D., LA to #115, M.D., UCLA, 2, 3/14/1955, *UCLA Hospital Records*, Folder U260. See also TJP, PER, 11/4/1954, *TJP Collection*, Box 21, Folder P178.

88. Letter, #102, M.D., Asst. Prof., Div. of Neurology, University of Minnesota Hospitals, Minneapolis, MN to Dr. #104, Minneapolis, MN, 2/10/1954, *UCLA Hospital Records*, Folder, P258.

89. Brickner, "Multiple Sclerosis," 743.

90. Schumacher, "M.S.," 1153.

91. #107, M.D, typescript sheet, 5/25/1955,*UCLA Hospital Records*, Folder U229.

92. #113, M.D. and #118, M.D., Physician's Notes, 5/2/1958, *UCLA Hospital Records*, Folder U238.

93. #105, M.D., Physician's Notes, 12/9/1958, *UCLA Hospital Records*, Folder U260.

94. #116, M.D., "Physician's Notes," 11/22/1960,*UCLA Hospital Records*, Folder U307.

95. TJP, PER, 11/10/1955, *TJP Collection*, Box 19, Folder P164.

96. See for example this 1966 letter from an official of the National Multiple Sclerosis Society to P80's husband in Beverly Hills:

> Dear <Husband of P80>: Thank you for your letter of January 18 and the enclosure on Dr. <name withheld>. Dr. <name withheld> has been treating patients for some seven or eight years with injections of an anti-inflammatory hormone, methylprednisolone, directly into the spinal canal. He accompanies the treatment with about a month of rehabilitative efforts consisting of active and passive movements, massage, etc. The injection of this hormone in this manner is practiced in a number of centers today including the Mayo Clinic in Rochester, Minnesota. As far as I know the others do not add the rehabilitative regime followed by <name withheld>. In none of these centers has controlled studies been done and we have only the impressions of the physicians so we are left without knowledge of the benefit that might be derived from this treatment. We cannot recommend the treatment- we can only tell you about it. We are always pleased to find dedicated men such as <name withheld> interested in and working with the multiple sclerosis patient. The important points to consider therefore are two: is the treatment harmful to the patient and are the charges exorbitant? Insofar as we know the use of this material is not harmful at the dosage used. I understand that a month at the Wilmington hospital costs about $1000 and at today's prices this is not exorbitant for thirty days of hospital

care. I would like to emphasize again that this is not a recommendation. We do no have proof of the ability of this treatment of modify the natural course of this disease," Letter, #16, M.D., Director of Medical Programs, National Multiple Sclerosis Society, NYC to P80's husband, BH, CA, 1/21/66, *TJP Collection*, Box 10, Folder P80.

Conclusion

The history of multiple sclerosis proved to be a useful probe in understanding the culture of health, medicine, and disease in the United States from the 1870s to the 1950s. As a National Multiple Sclerosis Society pamphlet put it in 1953: "the story of the knowledge of multiple sclerosis is like a history of medicine in miniature. The story of multiple sclerosis is not yet closed, but neither is the history of medicine."[1] This analysis has demonstrated that the story of multiple sclerosis frequently led off into different narratives. Understanding the history of multiple sclerosis required understanding these broader structures in which physicians and patients experienced this disease. The forces which explained the development of ideas and practices around multiple sclerosis emerged not from within the category "multiple sclerosis" but in the multiple cultural contexts in which the disease was embedded.

For example, the genesis of the category "multiple sclerosis" in France in the 1860s had to do with the particular institutional structure of the Salpêtrière and a particular style of medical practice which had evolved in Paris. In this local context, Charcot and others were constructing neurology as a specialty. Thus, the history of multiple sclerosis, in its very beginning, is inseparable from the history of neurology as a specialization. However, this study also has shown that what constituted neurological practice, what counted as neurology, depended on local conditions: that is, the neurologies in Germany, France, and the United States were different because of varying traditions of institutional structure, medical and national culture, and professional status. These different national and local contexts meant that the experience of physicians concerned with multiple sclerosis and patients who either received the diagnosis or suffered from undiagnosed demyelination and overgrowth of glial tissue, would be different depending on their particular locations.[2] Therefore, it is essential to study the experience of disease in local context.

For the particular environment of the United States one sees a gradual rise in the number of MS cases diagnosed from the late nineteenth century to the mid twentieth century when neurologists came to consider MS one of the most common diseases they saw. That MS was rare in 1900 but common in 1950 was not because there were more biological occurrences of MS; rather, the causes which explain the rise in MS cases can be found in several social sources: the ongoing construction of the specialty of neurology in the United States (a process which cannot be understood apart from the simultaneous construction of psychiatry and psychology)[3] meant that more diagnosticians had the training to "see" multiple sclerosis; the increased urbanization of the United States meant that more patients were in the field of vision of neurologists; and the decline of other disease categories, especially syphilis of the central nervous system and hysteria, meant that physicians expected to see more MS cases and so they did. The history of the diagnosis of MS also showed the crucial importance of analyzing individual diseases in the larger context of the ecology of disease in which particular maladies are embedded.

Likewise research interest in multiple sclerosis at the New York Neurological Institute in the 1920s and early 1930s was not due to the internal logic of a neurological laboratory's scientific findings; that is, the logic of scientific experiment did not drive the history of multiple sclerosis forward, not even its intellectual history; rather, studying multiple sclerosis served the institutional interests of the Neurological Institute in terms of building up their own laboratory facilities and in terms of advancing the research careers of individual neurologists. Moreover, the neurologists territorially marked multiple sclerosis along with epilepsy as diseases which were the property of the specialty of neurology. This was done at a time when the boundaries between neurology and her wealthy sibling psychiatry were still being negotiated. Basically, New York Neurological Institute neurologists used the money from the Commonwealth Fund, while ostensibly for research on multiple sclerosis, for personal and institutional goals.

In fact this multiple purposing proved to be a source of tension between the Commonwealth Fund and the New York Neurological Institute. This tension was endemic to the medical culture of the time and the lay culture of philanthropic entrepreneuralism. Though the New York Neurological Institute neurologists represented their research proposals in an elegant and unified way, the reality of their practices was that they engaged in highly autonomous and disconnected work in accordance with the norms of the medical culture of the day.[4] The cultural tradition of private voluntarism in the United States along with a period of economic prosperity created the conditions which made the financing of research on multiple sclerosis in New York in the 1920s possible.

This cultural tradition of voluntarism took the form, in one instance, of the National Multiple Sclerosis Society in the late 1940s. The emergence of this private group came at a time when there was a new cultural consensus in the United States about the role of government in medicine and disease. The coming together of these cultural forces produced a rapid increase in the amount of research on multiple sclerosis in the United States beginning in the late 1940s. Thus, the force for change in the science of multiple sclerosis came from the work of lay activists; and, more deeply, a particular cultural model of health and disease made possible and generated this lay activism. American neurologists welcomed this activity because they were in a relatively weak position as a specialty versus other medical specialties in the 1940s and 1950s.

Neurologists were able to harness the newly found millions of dollars available for research on multiple sclerosis from the late 1940s through the 1950s toward the service and consolidation of their specialty. They channelled the money raised by the National Multiple Sclerosis Society and the funds provided by the National Institute of Neurological Diseases and Blindness into what was virtually a single treasury. This served to nationalize the research program of multiple sclerosis and overcome the tradition of localism and individualism in medical research.

This nationalization did not occur in the therapeutic encounter between patient and physician. The highly decentralized political economy of clinical practice in the 1940s and 1950s made the conditions for diverse therapeutic practices with regard to MS possible. This political structure cannot, however, explain the attractiveness of the cultural model of the highly autonomous practitioner. Patients put enormous pressure on physicians to treat MS and this coincided with a medical culture which valued therapeutic activism. These practices made sense to physicians and patients in the context of their times. In other words, these therapeutic strategies "worked" within the logic of this particular medical cultural system.

A particular cultural and political formation created the conditions which made possible the therapeutic practices around multiple sclerosis. Thus, that which more powerfully explained practices around multiple sclerosis had less to do with the category "multiple sclerosis" itself but with the culture of health and medicine in which people experienced MS.

ENDNOTES

1. National Multiple Sclerosis Society, *Self Help* (Bethesda, MD: National Multiple Sclerosis Society, National Institute of Neurological Diseases and Blindness, 1953), 8.

2. I do not mean to suggest an "essentialist" concept of the disease multiple sclerosis. Concepts like multiple sclerosis require the mediation of language for even the simplest elucidation and are therefore inevitably constructed as soon as one writes, thinks, or talks about them. We have no meaningful access to the natural world except through language. Nevertheless, there is a sense in which a person experiences their bodily materiality in a way that is not simply reducible to language. The problem is that the only way one can talk, write, or think about this is through the mediation of language.

3. Jack Pressman, "Concepts of Mental Illness in the West," in the *Cambridge World History of Human Disease*, ed. K.F. Kiple (New York: Cambridge University Press, 1993), 59-84.

4. For this medical culture which "prized individual experience and judgment above all else," see Harry M. Marks, *The progress of experiment: Science and therapeutic reform in the United States, 1900-1990* (New York: Cambridge University Press, 1997), 53 and 46, 51, 109, 111, 113.

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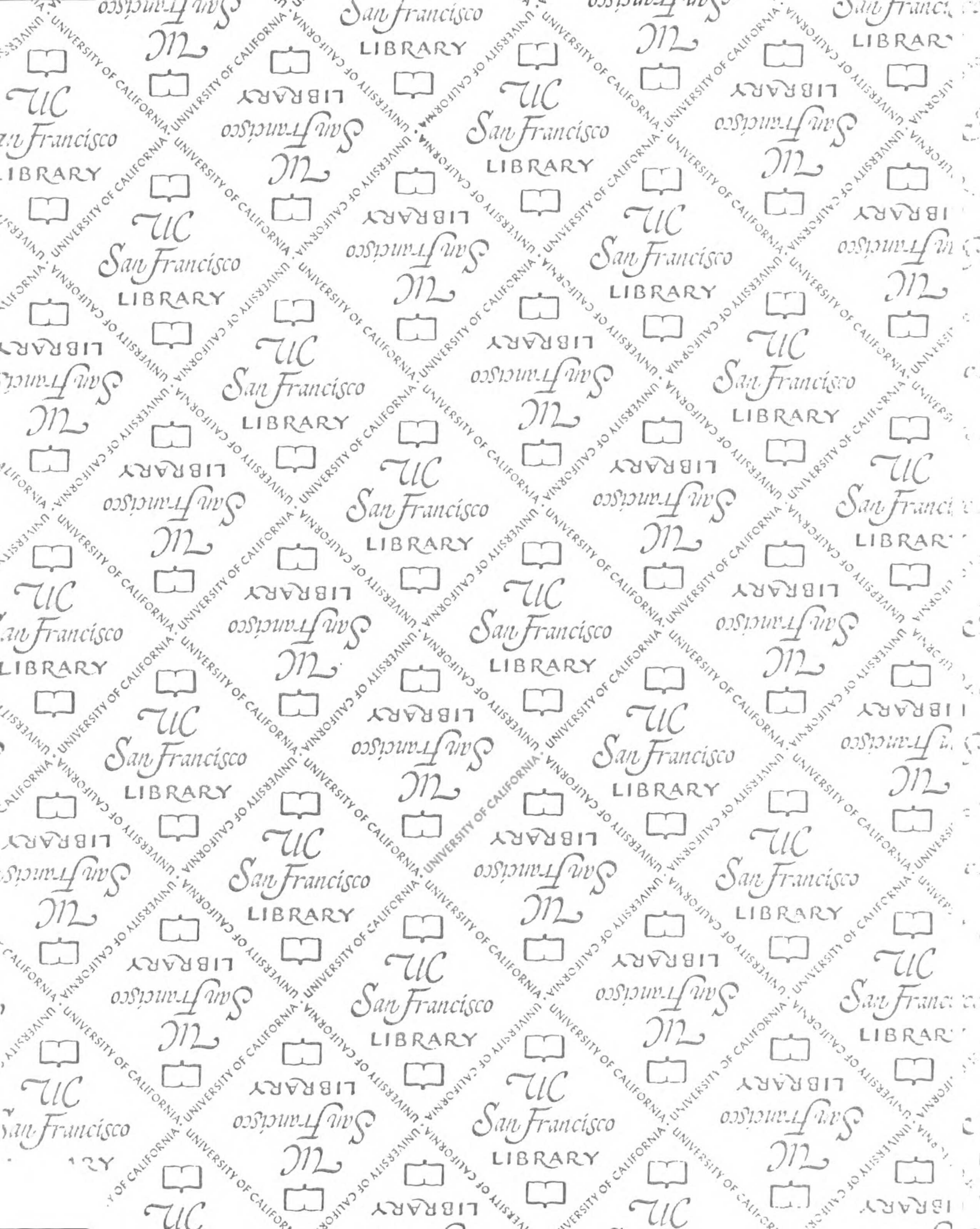
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