The First Modern Plague: Epidemic Encephalitis in America, 1919–39

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Epidemic encephalitis, which blossomed in the interwar period, came and went without anyone really understanding what it was. Shortly after Austrian neuroanatomist Constantin Von Ecomono described a new disease featuring flu-like symptoms, extreme lethargy, and eye paralysis in 1917, similar cases of "encephalitis lethargica" began appearing in the northeastern United States. Most investigators initially thought the disease was caused by a "filterable virus," but after two decades of research, the disease had mysteriously disappeared, and they had failed to isolate its causal micro-organism, leading some to suggest it had never been an infectious disease in the first place. My historical research attempts to reconstruct the neurological, epidemiological and bacteriological concepts, practices and institutions that nurtured epidemic encephalitis as a disease category, only to abandon it by the dawn of the Second World War.

New York City soon emerged as the focal point of the North American epidemics. The largest number of cases were found here, as was the greatest concentration of encephalitis research. Archival and published sources indicate that neurologists there embraced the disease as a unique opportunity to establish their hegemony over their European counterparts. Having lost poliomyelitis, syphilis, and neurasthenia to other medical specialties, New York neurologists hoped encephalitis would serve as a "model disease" through which they could extend their professional expertise into twentieth-century public health.

After renaming the disease "epidemic encephalitis," the New York researchers successfully created philanthropic and institutional support for their investigations. Basing their efforts out of the New York Academy of Medicine (NYAM) and the Neurological Institute of New York, they enjoyed generous support from William J. Matheson, a wealthy manufacturing chemist and scientific philanthropist. The "Matheson Commission" published four reports on epidemic encephalitis between 1929 and 1941, but came to few conclusions

regarding the true nature of the disease. The discovery of new "variants," such as Japanese B and St. Louis encephalitis, the aetiology of which was demonstrable by laboratory experiments, encouraged a new skepticism about the nature of epidemic encephalitis as a distinct disease entity. By the beginning of the Second World War, the investigation of the disease had been all but abandoned. With its sponsor dead, the Matheson Commission effectively concluded its study of epidemic encephalitis.

Archival sources held at the College of Physicians of Philadelphia (CPP) suggest a rather different response to the disease. They offer a more quotidian picture of epidemic encephalitis as a troubling new clinical problem, not as a potentially useful investigative model.

Admissions records of the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases from the 1920s and 1930s are terse, but nonetheless helpful. They provide rudimentary biographical data on patients, dates of admission and discharge, ward assignment, diagnosis, the name of the attending physician, and change in condition upon leaving the hospital. Philadelphia Orthopaedic was the oldest neurological hospital in the country, so it is no surprise to find that a substantial number of encephalitis patients went there for medical care. Between 1–3% of the 830–1,100 patients admitted each year were diagnosed with encephalitis or its sequelae. While epidemic encephalitis was officially a disease of winter and early spring (thus distinguishing it from the mosquito-borne versions that have recently captured the media's attention), patients often arrived in late summer or autumn, and they stayed for extended periods: three months was common, and the longest recorded stay was a few days short of a year. These patients undoubtedly provided a useful resource for teaching students and practitioners about this protean disease, which, neurologists insisted, was usually misdiagnosed as influenza, if its acute form wasn't ignored altogether.

Most patients paid for the privilege of diagnosis, and for what limited treatment the hospital might offer. The proportion of free patients tended to hover around 10%, which was the same proportion of free patients in the general hospital population at the time. Between 1920 and 1923, patients were diagnosed with "encephalitis" or (occasionally) "sleeping sickness," and were almost always discharged "cured" or at least "improved." Treatment relied on old standards: hyoscine or (in more serious cases) strychnine. But around 1924, the disease began to take on a chronic character, in keeping with the general tenor of American public health concerns at the time. Patients frequently received the diagnosis of "Encephalitis—P.A. type," or "postencephalitic Parkinsons." Interestingly, they were still

discharged with the same sparkling optimism about their improved condition, which continued to rely on the same old drugs. Very few patients died in hospital, despite the fact that the disease's mortality rate was estimated at anywhere between 10% and 30%.

There are hardly any cases of Parkinsonism in these records that are not attributed to encephalitis. This could indicate a decisive connection between the two, but it could just as easily be wishful thinking, as there was no definitive diagnostic test available. The casebooks of Charles Walts Burr (1861–1944), who saw encephalitis patients from 1928–1941 in the wards and at the out-patient clinic of Philadelphia Orthopaedic, indicate that many patients he diagnosed with encephalitis gave no history of an acute encephalitis, or even of influenza. Like many neurologists, Burr anticipated this connection when he interviewed his patients: he routinely recorded that the patient "denies acute encephalitis" or "denies influenza," almost as though he were seeing a tertiary syphilitic who "denied" having syphilis.

In fact, Burr frequently suspected syphilis was the true cause of his patients' degenerative nervous condition, and his casebooks indicate that he was particularly likely to suspect this if the patient in question happened to be black. H.U., for example, was a 22-year-old Philadelphia native who suffered a "nervous breakdown" in 1920. He complained of double vision, and was perpetually sleepy. He had had numerous Wassermann tests (most of which came back negative) before Burr saw him, but Burr insisted on ordering yet another test, presumably in an attempt to convince the man's father than the patient should immediately undergo fever therapy. B.C., a 32-year-old black chauffeur from Virginia, saw Burr in October of 1929. He reported no incidence of encephalitis or influenza, but did experience a "tremor of the head" while in the army in 1918. Now he walked with a stiff gait, his arms close by his sides. Burr immediately ordered a Wassermann, which came back negative.

Although Burr did order Wassermann tests in some of his *published* cases of white patients, his private casebooks do not offer similar examples. This perhaps reflects Burr's deep-seated and well-known eugenist opinions. In addition to serving as the first Chair of Psychiatry at the University of Pennsylvania (1901–30) and as the President of the American Neurological Association (1908), Burr had also recently been elected President of the National Eugenics Research Society (1926).

Burr argued racial differences were to blame for encephalitis's diverse clinical profile. A clinician thus had to understand the "soil" before he could sort out which symptoms were caused by an

infectious agent, and which were the result of racial degeneration. One of his patients, a 19-year-old German-Russian Jewish male, suffered from paralysis agitans as well as adolescent dementia. Burr described the mother as "neurotic" and "hysterical," while the father was "prone to outbursts of causeless anger" alternating with "periods of depression." The boy's movement disorder, Burr argued, was the product of encephalitis, but his dementia was surely part of his inheritance.

Burr, along with fellow neurologists Theodore Herman Weisenburg (1876–1934) and Francis Wharton Sinkler (1877–1954), oversaw the care of the majority of encephalitis patients at Philadelphia Orthopaedic. But, unlike their counterparts in New York, they never developed a coherent, institutionalized research program for studying the disease. At most, encephalitis in Philadelphia merely provided fodder for Burr's fears of racial degeneration. The only systematic studies of encephalitis in Philadelphia came not from neurologists, but from two psychiatrists, Earl D. Bond and Kenneth Appel. Both psychiatric reformers, Bond and Appel created a unique school for the re-education of post-encephalic children at the Pennsylvania Hospital in 1924, the results of which were published in 1931. Their report said nothing of racial degeneration, and focused instead on the social and economic status of their patients' families.

Eugenist interests were shared, although perhaps not so readily expressed, by neurologists in New York, just as they were in Philadelphia. So why did encephalitis develop differently as an object of neurological knowledge in the two cities? Local conditions certainly had an important effect here. Neurologists insisted that encephalitis was a new disease that usually went undiagnosed; its evaluation was very much a problem of information. If clinicians were unable to see a coherent disease beneath a myriad of bewildering symptoms, encephalitis went undiagnosed. Neurologists in New York enjoyed a remarkably strong network during this period that their Philadelphia counterparts seem to have lacked. New York neurologists had an intimate association with city and state public health authorities, and they repeatedly exploited this to their advantage, ultimately forging the Matheson Commission out of the NYAM's Commission on Public Health (itself run by a neurologist, Charles Loomis Dana). This gave them access to a huge number of records, the authority to coax physicians into submitting detailed patient records, and a mandate to launch a broad educational campaign. The CPP's Public Health Committee, in contrast, heard but two lectures on encephalitis during this period, both of which were delivered by Burr, who

seems to have already solved the mystery of the disease to his own satisfaction.

My thanks to the Resident Research Fellowships Review Committee at the Wood Institute for the award that made this research possible, and also to the expert assistance of Ed Morman and the staff at the CPP, who made my time there enjoyable & profitable.