Original Articles.

THE WORK OF THE MASSACHUSETTS STATE BOARD OF HEALTH IN THE INVESTIGATION OF INFANTILE PARALYSIS.*

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IN the year 1907 there occurred in New York City the most extensive epidemic of infantile paralysis of modern times. This epidemic was made the subject of an investigation by a committee of physicians representing various medical societies, and their results were later published.¹ In the fall of 1907 it became evident in Massachusetts that there were more cases occurring in the state than in previous years, and it was decided by the Massachusetts State Board of Health that an investigation into the distribution and characteristics of the disease as it appeared in the state over a period of years might be of value. In February, 1908, circular letters were sent out by the Board to every physician in Massachusetts—some 6,000 in all—asking if in the year 1907 they had seen in their practice any cases of acute febrile disturbance in young children, followed by paralysis. To the physicians replying in the affirmative, blanks, following in general those of the New York committee, were sent to be filled out, and the reports of 234 cases were received and analyzed.

These cases were distributed throughout the state in a general way corresponding to the density of population, although certain foci of increased incidence of the disease occurred in different parts of the state, such as Pittsfield and the valley of the Merrimac River. The environment and history of each case was recorded as far as possible, and it was announced that the Board would continue the investigation on the same lines in the following year. The data, along with a resumé of the subject, were published in the summer of 1908.²

In 1908³ the investigation was carried on along the same lines as in 1907. As at that time we had no direct proof that the disease was infectious, an attempt was made to investigate the stools of children sick with the disease on the ground that perhaps some especial bacteria might be found in the stools. Prof. Theobald Smith, pathologist to the Board, conducted this research, which proved negative, and was abandoned in 1909, after Flexner's demonstration that the disease was an infectious one.

In 1907 there occurred only 136 cases, and half of these occurred as an epidemic in Franklin County. The distribution of the cases was different from that of 1907, and communities affected one year apparently were comparatively immune the following year. The epidemic of 69 cases in Franklin County in 1907, just spoken of, was carefully studied by Dr. H. C. Emerson

of Springfield,³ at that time one of the medical inspectors of the Board, and in his conclusions he stated that the disease was evidently infectious, and at the most, mildly contagious. He also presented to the Board his conclusion that the disease was probably transmitted by insects, but this part of his paper was suppressed on the ground that the evidence was insufficient.

In 1909 the investigation showed 923 cases occurring in Massachusetts. Up to this year the work of collecting data had been done by mail by the clerical force of the Board, the blanks being filled out by the practitioners, but with the very marked increase in the cases, it seemed advisable to obtain more data and to secure them on the spot for the sake of uniformity, by a special investigator, and Mr. P. A. E. Sheppard, at that time a senior student in the Harvard Medical School, was engaged by the Board for this work, and continued in its employ until the autumn of 1912. It was his duty to visit as many cases as possible during or soon after the attack, and to fill out a blank embodying the information required.

In order to secure the best advice from various points of view in formulating the inquiry, four gentlemen were asked to serve as an Advisory Committee to the Secretary of the Board, Dr. M. W. Richardson, and to the writer who represented the Board in the investigation. These were Dr. Theobald Smith, Professor of Comparative Pathology; Dr. M. J. Rosenau, Professor of Preventive Medicine and Hygiene; Dr. J. H. Wright, Pathologist to the Massachusetts General Hospital and Assistant Professor of Pathology; and Dr. John Lovett Morse, Associate Professor of Pediatrics in the Harvard Medical School, and this Advisory Committee has continued to give its time and advice in the inquiry without compensation up to the present time, meeting as often as appeared necessary.

In November, 1909, the disease was made a notifiable one in Massachusetts, placing it in the category with scarlet fever, diphtheria and similar diseases. The attitude of the board in the matter at this time was expressed in the report for 1909, as follows: "It is only proper to state that in the present state of our knowledge no one can say which data are relevant and which are not. It is possible that some data which now seem of no importance may be of assistance to some future investigator."

In November, 1909,⁴ came the demonstration of Flexner and Lewis, that the disease was infectious, was due to a filterable virus, and could be produced in monkeys.

The routine data for 1909 were presented without conclusions, and were published as in former years.⁵ In the official publication⁶ of the Board covering 1909 were included (1) the general report for 1909; (2) an analysis of an epidemic of 82 cases in Berkshire County by Dr. L. A. Jones, Health Inspector of the Board for that district; (3) an article on the treatment of infantile paralysis by the Orthopedic Depart-

* Presented by invitation before the Academy of Arts and Sciences, Boston, January 8, 1913.

ment of the Harvard Medical School, previously published elsewhere,⁷ and (4) a paper on the diagnosis in the early and prodromal stages by W. P. Lucas, work done from the Proctor Fund of the Harvard Medical School.⁸

Up to and including the year 1909, the investigation had been carried on from the regular appropriation of the Board, but in January, 1910, the legislature was asked to appropriate \$5,000 for the continuation and extension of the study, now becoming more expensive. This money was voted without question and without opposition. In February, 1910, another investigator, Dr. J. P. Hennelly, a recent graduate of the Harvard Medical School, was detailed to the investigation of cases.

In 1910 there were reported in the state 845 cases, and a large epidemic of 148 cases occurred in Springfield, and another of 89 cases in Fall River. The publication for 1910 contained (1) the routine report of the disease for the year⁹; (2) a careful study of the Fall River epidemic by Dr. Hennelly¹⁰; (3) an extended analysis of the Springfield epidemic by Dr. Sheppard¹⁰; (4) an article by Rosenau, Sheppard and Amoss dealing with an unsuccessful attempt to produce the disease in monkeys by inoculation with the nasal, pharyngeal and buccal secretion of 18 human cases⁹; and (5) an investigation⁹ by Dr. May, a veterinary surgeon, who made at the request of the Board an inquiry into the occurrence in Massachusetts in 1910 of paralysis in the lower animals and fowls. Many cases of such paralysis were found, and the report was of interest, but seemed to throw no especial light on the relation of such paralysis to the human disease. In this year, as in previous ones, the Board bought paralyzed animals wherever obtainable, and sent them to Prof. Theobald Smith for investigation.

In 1911 the legislature was asked for \$10,000 for the extension of the inquiry, especially necessitated by the increasing size of the yearly publication and for other reasons to be spoken of. This amount was granted without opposition or delay.

Before passing to our analysis of the data collected in the four years ending in 1910, a brief resumé of the routine work for 1911 should be given. In that year 260 cases were reported, and certain work, not yet published, was done as to the possible relation of the disease to Herpes zoster.

In April, 1911, Dr. B. E. Wood of Boston, an orthopedic surgeon of experience, was requested to examine and report to the Board upon the condition then of the 234 cases paralyzed in 1907. He reported complete or functional recovery in 25 per cent. of these.¹¹

In December, 1911, Dr. W. P. Lucas and Dr. R. B. Osgood of Boston, undertook an inquiry to ascertain whether an immunity to various bacterial infections artificially produced in monkeys, produced also immunity to the virus of

infantile paralysis. The experiments seemed to show that in the Rhesus monkey such immunity afforded no demonstrable protection to the virus of infantile paralysis.¹²

A study of the blood in 13 acute cases of the disease was made in 1911 and reported to the Board by Hammond and Sheppard.¹³

We had now been at work for four years along definite and more or less unvarying lines, the collection of data covering points presumably of interest, and it seemed as if four years of such data was a sufficient number from which to draw conclusions, and that future work had better be formulated after a careful study of these conclusions and on the lines indicated by them, because the indefinite continuance of field work seemed to offer little promise of bringing out new facts. The data were therefore analyzed by Dr. Richardson and the writer.¹⁴

It was found that from 1907 to 1910 inclusive there had been reported to the board 2138 cases, and on the whole there seemed to be a two-year periodicity when limited localities were considered. In each of the years and taking all four together it was evidently an affection relatively more prevalent in the smaller cities and towns than in the larger, the average population in the 25 places where the disease was most prevalent being about 5000, and in the 25 where it was least prevalent being 52,000. This was not the case with scarlet fever analyzed as a control, which was relatively as prevalent in the same years in the larger cities and towns as in the smaller.

Bearing still further on the fact that infantile paralysis is essentially a rural disease, the number of domestic animals in proportion to the population was investigated in the 25 cities and towns where the disease was most prevalent and in the 25 where it was least prevalent. In those where the disease was least prevalent there was one cow to 84 inhabitants; where most prevalent one cow to 11 inhabitants; where least prevalent one horse to 32; where most prevalent one to 14. As to swine, there was one to 287 inhabitants where least prevalent; one to 60 where most. An investigation as to poultry showed one fowl to 22 inhabitants in the least affected regions; one fowl to 1.75 inhabitants in the most affected. Both analyses thus clearly brought out a fact established elsewhere,—that the disease was by preference rural rather than urban.

The seasonal occurrence was as usual, the disease reaching its height in either July, August or September in the various years, but never quite disappearing even in winter. The investigation of age, sex and mortality added nothing to the facts known. In the study of the environment of the patients in these four years there was no marked predilection of the disease for any especial floor in the house, and the age of the house, the sanitary condition and the degree of dampness seemed to be about the average in the community. The investigation as to the nearness of the house to the railroad track

showed nothing of apparent importance. An attempt was made to study the rôle of dust as a possible agent of the disease, and what was classed as an excessive amount was recorded in 36 per cent. of cases carefully studied. The amount of rainfall was also studied, which showed that the largest number of cases did not occur in the driest years.

The question of the possible transfer of the disease by insects and vermin had been tabulated from the first, and "flies" were naturally reported as present in 90 per cent. of cases and mosquitoes in over 40 per cent., but the information was not collected by an expert, and seemed to be too general to be of value.

The sickness, paralysis or death of domestic animals in infected families, was carefully recorded in connection with the cases and independently by Dr. May, but we could not feel that the number reported was necessarily larger than might have occurred in non-infected families, and the collection of control figures seemed hardly worth while, especially as Prof. Theobald Smith had inoculated monkeys with filtered emulsions of the cords of 12 such animals, the results of which were negative, and had been furnished with a fund raised by private subscription for an inquiry into animal paralysis and its possible bearing on the disease.

The patients affected were studied as to swimming, accidents and exposure just before the attack, as to previous diet and health, as to diseases prevalent in the town at the time, all of which seemed to show little of importance and to warrant no especial conclusions. The rest of the figures related to the clinical features of the attack, which had no apparent bearing on the etiology.

We found what appeared to be evidences of transmission by direct personal contact in 13 per cent. of cases carefully studied, and in 7 per cent. of the families more than one case occurred. Still, the evidence in favor of a contagious disease was not quite satisfactory, for institutions for children were practically immune, and in the one or two instances of disease in such institutions it did not spread, nor did it spread in schools or where children were crowded together. The distribution in general was erratic, isolated cases occurred, and the incubation was variable when it could be determined. Moreover, the attempts of Rosenau, Amoss and Sheppard, and of Strauss of New York, to transmit the disease by nasal and mouth secretions from persons in the acute stage of the disease had proved negative.

These data were then presented to our Advisory Board, and discussed with them. Considering all the facts, our attention was naturally turned to insects as a possible means of transmission. We had spoken of our suspicions in this direction in the 1909 report, and in the 1910 report expressed our belief that such an investigation to be of value must be made by an expert entomologist. After the study of the

data spoken of, it was decided to make such an investigation, and in the summer of 1911 Mr. Charles T. Brues, Instructor in Economic Entomology in Harvard University, was secured by the Board to go with the investigator, Dr. Sheppard, to see the acute cases and to report to the Board as to the prevalence of especial insects, especially biting insects, in these localities.

Between July 30, and September 26, 1911, the houses of 88 patients in 17 cities and towns were visited, and the stable fly was found in the environment in practically every case investigated. This and the mosquito were the only biting insects found constantly present. It seemed that if either of these insects were responsible it probably was the stable fly, as, if it were the mosquito, the disease would be more widespread, and would attack adults as well as children; whereas the fly would be more likely to bite children than adults, and the seasonal distribution was rather more like that of the fly than of the mosquito.¹⁵

Prof. Rosenau was then requested on behalf of the Board to undertake the experiment of transmitting the disease from monkey to monkey by means of the stable fly, and with Mr. Brues reported at the Congress of Hygiene in Washington on September 26, 1912,¹⁶ that inoculated monkeys had been bitten by stable flies, and that of 12 healthy monkeys subsequently bitten by the same flies six showed indications of the disease. This was subsequently confirmed by independent experiments of Anderson and Frost.¹⁷

The sequence was then as follows: (1) the routine collection of data for four years, with the publication of these data at the close of each year without theorizing; (2) an analysis and study of these data at the end of four years; (3) deductions from these data; (4) new lines of observation founded on these deductions, leading to (5) Rosenau's investigations and discovery.

If I have dwelt somewhat insistently on the part played by the State Board of Health in this matter, somewhat more than may have seemed wholly necessary, it has been because I am anxious to lay stress on the fact that this combined work between a public health board and the scientific men of a university medical school seems to offer possibilities of much use in the development of preventive medicine. To speak of this desirable combination as a theoretical matter is one thing; to point to a practical achievement brought about by this team work is another.

In the investigation of a public health problem of this sort most medical schools have neither the funds, the clinical material, nor the clerical force necessary for a long continued investigation. On the other hand, the public health boards do not as a rule have scientists of the first rank who are free to work on the purely scientific side of such problems. The

public health boards, however, have immense clinical material, for they are in touch with a large number of physicians and they have machinery for the assembling of desirable data.

Our own experience in the latter respect bears directly on the question. We have found the medical profession of the state interested, in sympathy with our efforts, and most helpful in every way almost without exception. The families of the cases investigated have given us every possible assistance, and have welcomed the inquiry. The legislature has been most liberal, and has granted us in all \$20,000 in three separate years, without question. With a profession interested and sympathetic, a public ready to help, and a legislature ready to back such work financially, the Massachusetts experience points to the advisability of such concerted investigation and research between public health boards and the scientific departments of medical schools.

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SHOCK. A REVIEW OF THE THEORIES AND EXPERIMENTAL DATA TO DATE.

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THE phenomenon known as shock was a condition recognized by careful observers as early as 1568, when Clowes, and later in 1719, Weismann, spoke of it as a condition probably caused by the presence of a foreign body in the wound or blood. A little later than this the condition was attributed to a disturbed molecular condition in the tissue.

From a purely historical point of view the literature devoted to shock may be divided into three periods:

(1) From 1795, when the word was first used as we now use it, down to the beginning of the era of experimentation in 1876.

(2) The period from 1870 to 1885, devoted to careful experimental investigation, and

(3) From the re-opening of the question by Crile in 1889, to the present time.

From 1885 to 1889 practically no papers, others than purely casuistic ones, appeared on the subject.

Nearly half a century before Crile's work, Keen, Mitchell and Moorehouse advanced the theory of vasomotor exhaustion, and experimental work by Fischer in 1870 seemed to confirm this theory, i.e. of a vasomotor paralysis with flooding of the splanchnic veins and contraction of the peripheral vascular system. This was the recognized phenomena but no adequate explanation of its physiology was at hand.

Brown-Sequard believed the condition to be one of central anemia due to persistent contraction of the capillaries throughout the vasomotor centers.

Moulin: A reflex paralysis in the strictest sense of the word; a reflex inhibition, probably affecting all the functions of the nervous system and not being limited to the heart and blood vessels only.

Boise: Hyperirritation of the entire sympathetic system with resulting stimulation of vasomotor nerves, contraction of arterioles and spasmodic heart action.

Kinneman emphasizes the rôle of a fall in temperature as the principal causative factor in shock, and as a result of extensive animal experimentation, finds that shock must not be considered as due to the lowering or exhausting of any one bodily function, but as a composite condition embracing an interference with normal height of the blood-pressure, interference with the respiratory act and a marked fall in body temperature. Of these, as shock progresses, the most uniform and progressive factor is the fall in temperature. Dogs, after shock, were immersed in a bath of saline at body temperature, and others in a bath of higher temperature. These experiments showed (a) that a sufficient fall in body temperature can cause a decrease in respiratory rate and a marked fall in blood-pressure, which in itself constitutes a shock; (b) conversely, a limiting of the fall of temperature limits the fall in pressure and prevents a fall in respiration. (c) On the other hand, a rise in temperature causes a rise in blood pressure and respiratory rate, reduced by shock, with the result of a gradual annihilation of all symptoms. Thus, of the three factors, temperature commands first place by its power of production, of limitation and amelioration of the composite condition—shock.

As the problem stands today there are several contradictory theories pretty well based on rather extensive experimental evidence. It is to the proper interpretation of this accumulated experimental data that we must look for the physiologic cause rather than to the theory or opinion of any one worker. It is interesting, and at the same time puzzling, to note that each observer is equally sure that he has discovered, through experimentation, the fundamental or chief underlying factor. Thus Crile maintains that vasomotor exhaustion is the primary cause. Boise states that cardiac exhaustion is the prime factor, whereas Howell believes that both cardiac