ape was allowed to drink a heavy suspension of freshly isolated Bordet bacilli. After an incubation period of twenty-four days, this ape developed typical whooping cough. The cough was accompanied by a marked leukocytosis and an absolute increase in the lymphocytes. Cough plates were positive for Bordet bacilli. The cough endured for six weeks. A third ape, which was inoculated with third generation cultures of Bordet bacilli isolated from the previous ape, developed typical whooping cough after an incubation period of seven days. Again, the cough was accompanied by a leukocytosis which at its height showed 63,000 white blood cells with an absolute increase in the lymphocytes to 44,730 cells. Cough plates were covered with Bordet bacilli. The cough persisted for six weeks. In all three apes, the complement-fixation reaction with pertussis antigen, which was negative during the incubation period, became strongly positive after the disease was well developed. Berkefeld W. filtrates of the rhinopharyngeal washings taken during the catarrhal stage in two of these three apes and inoculated into other apes did not produce catarrhal affections.

The interpretation of these observations is difficult. There can be no doubt but that the three apes inoculated with pure cultures of Bordet bacilli or whole sputum developed typical whooping cough, which in its clinical and bacteriological aspects was indistinguishable from the human disease. However, the results in those apes inoculated with filtrates from human cases are inconclusive and are open to two interpretations. One, that the catarrhal affections represented the action of a hitherto unrecognized filterable virus which acts as the primary infecting agent in whooping cough, or that the catarrhal affections were simply common colds and that we were dealing with whooping cough cases who were also carriers of the virus of the common cold. Further investigations are being made upon all aspects of the experimental disease.

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AN ENCEPHALOMYELITIS IN THE CHICKEN[1]

A NERVOUS disorder of chickens, in the form of a pronounced and rapid tremor of the head and neck with which, in some cases, there is also an associated ataxia, was first seen by us in May, 1930, when a group of nine diseased chickens was brought to the laboratory. In April, 1931, a second group of affected birds was received, a third in January, 1932, and a fourth in May, 1932. Throughout the spring of 1932 reports of the wide-spread occurrence of this disease have been frequent.

In an affected flock, the first symptom of the disease to be observed is the rapid tremor of the head and neck in certain birds. On manual examination, other muscles are found to be involved as well. The "trembling" becomes aggravated when the chickens are excited, tends to subside when the birds are left to themselves, and disappears in sleep. In some chickens the tremor is very fine, in others coarse; in some it is intermittent in character, in others it appears to be constant during their waking hours.

An ataxia was present in addition to the tremor in some of the birds when they were brought to the laboratory, and developed in others while they were under observation. An occasional bird, showing the tremor only, gradually recovered, and at the end of two to three months appeared to be normal; others continued to show the tremor over the same period; but those affected with an ataxia in addition to the tremor grew progressively worse, and sometimes within a week after onset of the ataxia were moribund.

The age of onset of the disease has been reported to be as early as two days after the chickens had been removed from the incubator. The youngest birds brought to us were five days old when we received them, the oldest, six to seven weeks.

A total of 102 affected birds have been studied. Detailed pathological examination of fifty of these has been made at the present time. Over thirty of the birds are still alive and are being used for further experiments.

Work with the disease has included experiments on production, transmission and cure. All efforts to produce the disease *de novo, i.e.,* by diet, high temperature, etc., have been unsuccessful. Transmission experiments, in which a suspension in salt solution of brain or of spinal cord of an affected bird was inoculated intracranially into very young normal chickens, have been successful in six cases. Typical disease with marked tremor and poor sense of balance was produced in four chickens; with marked tremor and pronounced ataxia in one chicken; and an atypical form with ataxia and no tremor was produced in one chicken.[2] The first two successful inoculations were made from suspensions of the cord from two chickens in the group brought to the laboratory in January, 1932. Inoculations with a brain suspension in each of these two cases were negative. The first

[1] Preliminary report.

[2] The lesions of the brain and spinal cord of this chicken were numerous and large. They were indistinguishable from lesions occurring in chickens with typical manifestations of the disease, *i.e.,* both tremor and ataxia.

inoculated bird to show the disease was chloroformed the day following that on which positive diagnosis of disease was made, symptoms having been observed two days earlier, twenty-eight days after inoculation. When killed, the tremor was pronounced, and the right leg somewhat ataxic. A suspension of the brain of this bird was made in salt solution and inoculated intracranially into each of four birds, and a suspension of cord was likewise inoculated into four birds. None of these had developed the disease at the end of six weeks when they were killed. Autopsies were performed and material for pathological examination saved in all cases.

The second inoculated bird to show the disease developed a tremor twenty-nine days after inoculation and was killed seven days later. A suspension of brain from this bird was inoculated intracranially into each of ten chickens, and a suspension of cord similarly into each of eleven chickens. None of those inoculated with the suspension of spinal cord developed the disease, but of those inoculated with the suspension of brain, one developed a distinct ataxia after twenty-eight days, and a second developed a marked tremor and poor sense of balance but no real ataxia after twenty-nine days.

These birds were chloroformed, one six days after the tremor appeared, the other five days, and a suspension of brain or of cord was inoculated intracranially into each of sixteen birds. One of the birds inoculated with cord suspension from the bird with tremor and no ataxia developed a fine rapid tremor thirty-four days after inoculation. Forty days after inoculation one of those inoculated with cord suspension from the bird showing ataxia and no tremor showed a fine tremor. Further inoculations are being made from the brain and the cord of each of these two chickens.

All the birds developing the disease after inoculation exhibited numerous lesions in both brain and cord. These lesions are typical of the disease and closely resemble the early lesions found in naturally affected birds. The inoculation experiments are being continued.

No method of curing the disorder has been discovered. In rare instances, birds have apparently "outgrown" the disease, but in the majority of cases either the tremor continues unchanged in intensity, or an ataxia develops which becomes progressively more severe until the bird is incapacitated.

The pathology[3] of the disease is not confined to the brain and spinal cord, although the lesions found there are constant in occurrence and type, while those occurring in the viscera vary in size and distribution. The lesions in the brain and spinal cord consist of focal collections of neuroglia cells around capillaries, perivascular infiltrations and degeneration of nerve cells. The neuroglia cells comprising the focal accumulations are chiefly macroglia and oligodendroglia, with an occasional microglia cell. Mitotic figures are frequently seen, indicating the proliferative nature of the lesion. These focal collections of glia cells are found in all parts of the brain and spinal cord, although fewer are found in the cerebellum than elsewhere.

The lesions in the viscera consist of microscopic areas of infiltration with lymphoid cells. These areas are either rounded and sharply circumscribed, or irregular in outline. Many of the cells, especially in the circumscribed areas, resemble lymphoblasts. Mitotic figures are often found, and also many pycnotic nuclei. The pancreas and heart are the organs most severely affected by these infiltrations, but liver, spleen, kidney, testis and other organs are also involved in varying degrees.

Further experiments on production and transmission of the disease are now in progress, and pathological studies are being continued. A complete account of the work will be published at a later date.

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[3] The pathology of the brain and spinal cord has been worked out by Dr. Myrtelle M. Canavan, of the Department of Pathology of the Harvard University Medical School. Dr. E. E. Tyzzer, Dr. S. B. Wolbach and Dr. Stanley Cobb have also examined many of the sections.

BOOKS RECEIVED

BARBOUR, HENRY G. *Experimental Pharmacology and Toxicology.* Pp. 141. 14 figures. Lea and Febiger. $2.75.

BEAN, ROBERT B. *The Races of Man.* Pp. iv + 134. 69 figures. University Society, New York.

HARRIS, J. ALLEN and WILLIAM URE. *Experimental Chemistry for Colleges.* Pp. x + 192. 21 figures. McGraw-Hill. $1.25.

MAGOUN, A. ALEXANDER. *Problems in Human Engineering.* Pp. xxxiv + 535. Macmillan. $2.60.

MINCKLEY, LOREN S. *Americanization through Education.* Pp. 107. Christopher Publishing House. $1.50.

NEELLEY, JOHN H. and JOSHUA I. TRACEY. *Differential and Integral Calculus.* Pp. viii + 496. 238 figures. Macmillan. $4.00.

OTIS, CHARLES H. *Michigan Trees.* Pp. 362. Illustrated. University of Michigan.

REEDY, J. H. *Elementary Qualitative Analysis.* Second Edition. Pp. x + 163. 13 figures. McGraw-Hill. $1.50.

SANSOME, F. W. and J. PHILIP. *Recent Advances in Plant Genetics.* Pp. x + 414. 56 illustrations. Blakiston. $4.00.

SINNOTT, EDMUND W. and L. C. DUNN. *Principles of Genetics.* Second Edition. Pp. xvi + 44. 168 figures. Tables. McGraw-Hill. $3.50.

TANAKA, SHIGHEO. *Japanese Fishes.* Pp. 203. Illustrated. Imperial University, Tokyo.

TITCHMARSH, E. C. *The Theory of Functions.* Pp. x + 454. Oxford University Press. $7.50.