

tobacco laboratory at Oxford, N. C., and from \$182,600 to \$191,000 for insects affecting man and animals. The decreases are distributed among seven projects, the largest being the control of screw-worms, Japanese beetles and gipsy and browntail moths, the insecticide and fungicide investigations and the insect pest survey.

The total for the Bureau of Plant Industry shows a decrease from \$4,909,048 to \$4,741,675, distributed among 12 items, the largest reductions being \$106,200 for sugar plant investigations, \$67,413 for the National Arboretum and \$64,750 for the studies of dry-land agriculture. These are offset in part by an increase of \$119,700 for studies with fruit and vegetable crops, particularly tung-oil investigations. The latter will be supplemented by chemical studies under an allotment of \$30,000 to the Bureau of Chemistry and Soils. This bureau is also given an increase of \$39,957 for work in the industrial utilization of farm products and by-products, especially sweetpotato drying and citrus byproducts. Despite slight reductions on its other projects, the total for the Bureau of Chemistry and Soils rises from \$1,425,431 to \$1,457,508.

The Bureau of Biological Survey is granted an allot-

ment of \$1,000,000 under a 1937 act for federal aid to the states in wildlife restoration projects, as well as \$25,000 additional for the development of fur resources investigations. Its total appropriation is increased from \$2,127,840 to \$3,248,340.

The remaining work of the department is provided for much as at present. The Weather Bureau receives \$4,987,870, a net increase of \$284,821, available entirely for additional aerological work; the Bureau of Dairy Industry \$717,405, an increase of \$13,711 for investigations; the Bureau of Home Economics \$305,085, an increase of \$60,000 to be divided equally between investigations of family economics and cotton hosiery; the Commodity Exchange Administration \$635,000, an increase of \$135,000; and the Bureau of Agricultural Engineering \$510,000, an increase of \$49,231. An apparent increase of \$220,640 to the Office of Information raises its total to \$1,474,770, but it is made up entirely of transfers from the Soil Conservation Service and provides no additional funds. The Food and Drug Administration again receives \$2,227,758, the Library \$105,420, and the Beltsville Research Center \$85,000 (an increase of \$10,000).

SPECIAL ARTICLES

RECOVERY OF THE VIRUS OF EQUINE EN- CEPHALOMYELITIS FROM THE BRAIN OF A CHILD

ON August 31, 1938, brain material from a twenty-months-old boy was received through the courtesy of Dr. H. M. Ginsburg, Dr. Newell and Dr. S. Simon, of the Fresno County General Hospital, Fresno, California. The child was admitted to the hospital on August 27 after a two-day illness. He became progressively worse, developed muscle spasms, became comatose and died on August 30, or 5 days after the initial onset, with the diagnosis of acute encephalitis.

A portion of the brain stem was ground in Ringer's solution to make a 10 per cent. suspension and was injected intracerebrally, intraperitoneally and intranasally into young Swiss mice. Most of them died in 3 to 5 days, while the others were sacrificed upon showing neurological symptoms; tremor, initial excitability followed by drowsiness and later complete prostration. The brains were removed and were found to be free from bacteria. A 10 per cent. suspension was inoculated into normal mice, who succumbed with similar symptoms. Berkefeld N filtrates were also infectious for mice, and it was found that the active agent could be carried in serial passage in these animals.

Mouse brain suspensions were then inoculated intracerebrally into monkeys, guinea pigs, young rabbits and rats. All developed typical encephalitic symptoms

of fever, tremor, twitchings of the extremities, drowsiness, spastic convulsions in the case of the monkeys, and became completely prostrate in 4 to 5 days. Spinal fluid of a monkey removed at the height of the temperature, 40.7° C, contained the virus upon inoculation of mice. Brain material from this monkey was infectious upon removal post mortem. The disease could be transmitted by the intranasal and the subcutaneous routes of inoculation in both mice and guinea pigs.

From the incubation period of 4 to 5 days, from the general clinical picture, especially in the guinea pig and from the fact that the virus could be transmitted to such a variety of animals by various routes of inoculation, the virus of equine encephalomyelitis, western type, was suspected. Further confirmation was obtained by neutralization, complement fixation and cross-immunity tests. Using methods previously described,¹ it was found that this new (Br) virus neutralized hyperimmune serums of the western type of equine encephalomyelitis but not those of the eastern nor the Moscow No. 2, nor was there any neutralization with immune serums of the viruses of lymphocytic choriomeningitis, the Japanese B nor the St. Louis types of human encephalitis, respectively. Guinea pigs immune to the western variety were unaffected by an intracerebral inoculation of the new strain, while two monkeys immune to poliomyelitis

¹ B. F. Howitt, *Jour. Immunol.*, 33: 235, 1937.

succumbed to the disease in 4 days. Guinea pigs immune to the virus of lymphocytic choriomeningitis and mice immune to the St. Louis strain were all susceptible to intracerebral injection of the Br virus.

Mouse brain antigens, prepared with this virus by methods previously described,¹ gave a positive complement fixation against immune serums of the western type but not against those of the eastern or the Moscow No. 2 equine strains nor of lymphocytic choriomeningitis.

During the summer months of both 1937 and 1938 numerous cases of encephalitis of unknown etiology have been reported from the San Joaquin Valley regions in California; especially from around Fresno and Tulare. Until the present time no virus had been recovered from the few specimens examined, but it had been shown² that serums from 56.6 per cent. of the recovered cases in this area gave neutralizing antibodies to the virus of St. Louis encephalitis. Although no virus was found, it was assumed that this strain was present in the locality. However, the clinicians were recognizing several different clinical pictures in their patients; an extremely acute, fulminating form with death in a few days, a lethargic type with a prolonged course usually with residual effects, and others that were milder, sometimes transient, ending in good recovery. Cases were reported in all age groups, with many in children and babies. Definite convulsions were often noted in the young children, persisting for some time. Serums from this latter group and from those having residual after-effects were usually negative when tested for neutralization against the St. Louis virus. At present, serums from three children having the convulsive type at the Kern County Hospital have been found to neutralize the new Br strain of virus and not that of the St. Louis. It seems probable, therefore, that both these viruses are responsible for cases of encephalitis in these regions.

Although the virus of equine encephalomyelitis was first reported by Meyer, Haring and Howitt³ from the horses in the San Joaquin Valley area in 1931, yet this is now the first time the virus has been recovered from a human brain in California. However, that it very likely could be transmitted to the human host from the equine was early considered by Meyer,⁴ who in 1932 reported one fatal and one non-fatal case of suspected encephalomyelitis in two men associated with sick horses. The symptoms were similar to those shown by many of the present cases. That the virus could be recovered from a human being had also been noted by the writer in 1934 when it was found in the cerebrospinal fluid of a man who died under such

unusual conditions that the case could not be reported at the time.

Although the equine disease has been endemic among the horses in the valley regions for a long period, yet there have been no unusual number of cases reported among the animals during the past few years.

It is interesting that the virus of equine encephalomyelitis should have been found in man almost coincidentally in two widely separated portions of the United States, being first reported recently from Massachusetts by Fothergill, Dingle, Farber and Connerly⁵ and by Webster and Wright⁶ for the eastern type and now in California for the western form, which is serologically and immunologically distinct from the other.⁷

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SIZE AND STROKE OF THE HEART IN YOUNG MEN IN RELATION TO ATHLETIC ACTIVITY¹

THERE is a common belief that strenuous athletic activity results in an increased size of the heart. It is also frequently considered that this may be a deleterious adaptation, either because it indicates undue strain or because of the danger of eventual degeneration. The scientific debate on this question is now more than thirty years old,² but there are important inconsistencies in the published data. Papers up to 1933 were reviewed by Steinhaus.³ In a more recent paper Roesler⁴ states that the diastolic outline was unusually large in four athletes and that the outline was decreased after cessation of training.

Our comment on previous work may be summarized briefly: (1) Most of the studies have been made with the orthodiagraph, so that the heart shadow is only outlined in diastole. (2) True resting conditions have not always been maintained. (3) When the teleroentgenographic method was used precise correction for distortion was not made from lateral roentgenograms on the different individuals. (4) Strictly comparable controls were not measured under the same conditions.

⁵ L. D. Fothergill, J. H. Dingle, S. Farber and M. L. Connerly, *New England Jour. Med.*, 219: 411, 1938.

⁶ L. T. Webster and F. H. Wright, *SCIENCE*, 88: 305, 1938.

⁷ Appreciation is extended to Dr. H. M. Ginsburg, Dr. F. Cooley, Dr. Newell and Dr. S. Simon, of the Fresno County General Hospital, and to Dr. M. A. Gifford and Dr. W. C. Buss, of the Kern County Hospital, for their kind cooperation and many courtesies offered.

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² Schieffer, *Deut. Arch. Klin. Med.*, 80: 604, 1907.

³ A. H. Steinhaus, *Physiol. Rev.*, 13: 103-147, 1933.

⁴ H. Roesler, *Am. Jour. Roentgenology*, 36: 849-853, 1936.

² B. F. Howitt, *Jour. Bacter.*, 36: 294, 1938.

³ K. F. Meyer, C. M. Haring and B. F. Howitt, *SCIENCE*, 44: 227, 1931.

⁴ K. F. Meyer, *Ann. Int. Med.*, 6: 645, 1932.