denervation causes death. These disturbances can be overcome and death avoided by destroying the hearts in 2 stages separated by an interval of at least a week which gives time for the development of new collateral pathways. The existence of a single intact lymph heart prevents the development of all disturbances, including death. As a result of the destruction of the lymph hearts, the lymph which transudes from the blood vessels collects in the lymph spaces and cannot return to the blood stream. Water, salts, and proteins coming from blood and tissues remain in the lymph. Water in large amounts comes from the external environment. The decrease in the content of water, salts, and possibly protein in the blood and the changes in the chemical composition of the tissues cause the death of the animal.

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Transmission of St. Louis Encephalitis to the Hamster.

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The virus of St. Louis encephalitis has been found to be pathogenic for relatively few laboratory animals. Muckenfuss, Armstrong and McCordock[1] first established the virus in monkeys, but it was difficult to maintain in this animal. Webster and Fite[2] showed that Swiss mice could be infected easily by intracerebral and intranasal inoculation. All types of albino mice as well as house mice,[3] field mice and meadow mice[4] are susceptible.

Smith[5] has shown that the virus of St. Louis encephalitis persists in the brain of rats and guinea pigs for 8 or 9 days after intracerebral inoculation and slight anatomical lesions are demonstrable. These animals, however, showed no symptoms of disease. The

1 Muckenfuss, R. S., Armstrong, C., and McCordock, H. A., *Pub. Health Rep.*, 1933, **48**, 1341.

2 Webster, L. T., and Fite, G. L., *Science*, 1933, **78**, 463.

3 Harford, C. G., Sulkin, S. E., and Bronfenbrenner, J., PROC. SOC. EXP. BIOL. AND MED., 1939, **41**, 331.

4 Greutter, J. E., Fulton, J. D., Muether, R. O., Hanss, E. V., and Broun, G. O., PROC. SOC. EXP. BIOL. AND MED., 1940, **44**, 253.

5 Smith, M. G., *J. Infect. Dis.*, 1939, **64**, 307.

virus is demonstrable in rat and guinea pig brain only after the first transfer. She concludes that the virus has not been established in mice and guinea pigs by serial passage. Sulkin, Harford and Bronfenbrenner[6] carried the virus of St. Louis encephalitis through 6 successive passages in guinea pigs and the following year through 5 successful passages. However, later they were unsuccessful in carrying the virus in this animal. Our own experience with inoculations of rats and guinea pigs is similar to that of Smith.[5] We have had on a few occasions death of inoculated rats on the sixth or seventh day after inoculation and were able to recover the virus from the brains of these animals. However, most animals proved resistant and serial transfer of virus from animal to animal was not successful.

Since studies of the serological aspects of St. Louis encephalitis would be facilitated by the discovery of a susceptible animal of somewhat larger size than the mouse, we have recently tried the effect of inoculation of the virus into the golden or Syrian hamster, *Mesocricetus auratus.*

This animal is only slightly smaller than the guinea pig. It can be readily propagated for laboratory use. Blood can be withdrawn from the heart in sufficient quantities to permit serological studies of individual animals.

We immediately found that this animal is highly susceptible to infection with the virus of St. Louis encephalitis both by intracerebral and intranasal inoculation. The animals show evidences of illness, such as weakness, somnolence and tremors on the third and fourth days after intracerebral inoculation. Usually within 24 hours after appearance of such symptoms the animal dies. Microscopic evidence of encephalitis is found on pathological examination of the brain. The presence of the virus can be shown by virus neutralization tests. Its concentration is very close to that found in the brains of mice.

After intranasal inoculation illness occurs on the fifth or sixth day with death taking place approximately one day later. Subcutaneous inoculation is not followed by evidence of illness. The results of oral administration are under study.

The virus has been carried through 4 serial transfers from animal to animal without loss of virulence.

We already have evidence that neutralizing antibodies may be demonstrated in the blood of these animals. Their time of appearance under various circumstances is now under investigation.

[6] Sulkin, S. E., Harford, C. G., and Bronfenbrenner, J., PROC. SOC. EXP. BIOL. AND MED., 1939, **41**, 329.

Summary. The Syrian hamster is highly susceptible to intracerebral and intranasal inoculation with the virus of St. Louis encephalitis.

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Anemia in Vitamin C Deficiency and Its Response to Iron.

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Experimental scurvy in the guinea pig is usually associated with anemia,[1] and antiscorbutic food[2] or vitamin C[3] improves the anemia together with the scurvy. In human nutrition, hypochromic anemia frequently accompanies scurvy in infants,[4] and it is also commonly found in adults with vitamin C deficiency. Mettier, Minot and Townsend[5] postulate that vitamin C exerts a specific effect on erythropoiesis. Dunlop and Scarborough[6] reported 2 cases of scurvy with anemia which disappeared on supplementing a deficient diet with 60 mg of ascorbic acid daily. On the other hand, Keefer and Yang[7] state that in man scurvy may exist without anemia, although, if the disease is of considerable duration and associated with hemorrhage, infection or general undernutrition, anemia may be present. Likewise, Abt and Farmer[8] feel that when anemia accompanies lack of vitamin C, it is probably due to a generally deficient diet in which substances other than vitamin C, especially iron, are lacking. The recent experimental production of scurvy in a human subject without appearance of anemia[9] also supports the latter viewpoint.

In view of the existing controversy in regard to the rôle of vitamin C in anemia, the following data obtained in the Spring of

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2 Mettier, S. R., and Chew, W. B., *J. Exp. Med.*, 1932, **55**, 971.

3 Aron, H. C. S., *J. Nutrition*, 1939, **18**, 375.

4 Hess, A. F., *Scurvy, Past and Present*, Philadelphia, Lippincott Co., 1920; Parsons, L. G., and Hawksley, J. C., *Arch. Dis. Child.*, 1933, **8**, 117.

5 Mettier, S. R., Minot, G. R., and Townsend, W. C., *J. Am. Med. Assn.*, 1930, **95**, 1089.

6 Dunlop, D. M., and Scarborough, H., *Edinburgh Med. J.*, 1935, **42**, 476.

7 Keefer, C. S., and Yang, C. S., *Nat. Med. J. China*, 1929, **15**, 419.

8 Abt, A. F., and Farmer, C. J., *J. Am. Med. Assn.*, 1938, **111**, 1555.

9 Crandon, J. H., Lund, C. C., and Dill, D. B., *New England J. Med.*, 1940, **223**, 354.