western equine viruses were performed with the sera of equine and human recovered cases and their contacts and also with sera from domestic animals found on the farms of fatal cases. The results are given in the Table I. Besides the positive results with the human and horse sera only 2 chickens and 1 dog showed strong antibodies for the eastern virus. The sera of 2 chickens, 1 goose, 1 pigeon and 2 cows were only weakly positive. Many of these tests were performed with one of the newly isolated human strains of virus. It was of interest that 25.9% of the unvaccinated horses, 18.1% of the chickens and 1 dog had antibodies for the western equine virus, while the sera of 2 cows and 2 people were weakly positive.

The histopathology in the horse brains showed focal and diffuse gliosis, perivascular round-cell infiltration and edema. The motor neurons showed chromatolysis with cytolysis and phagocytosis by the glial cells. Passage of a human strain into guinea pigs produced severe neuron degeneration and mild perivascular infiltration with edema.

Arthropods were collected from the different areas where encephalitic cases occurred. A later report will be presented as well as a more detailed epidemiological account of this outbreak. The clinical cases will be described by Dr. J. Syverton of Louisiana State University.

*Summary.* The virus of eastern equine encephalomyelitis was recovered from the brains of both human and equine fatal cases occurring in an outbreak of encephalomyelitis in southwestern Louisiana. Positive neutralization tests to this virus were obtained with sera of 2 chickens, 1 dog, and 31 human and equine cases and their contacts, while those of 2 chickens, 1 goose, 1 pigeon and 2 cows were only weakly positive. Antibodies to the western equine virus were found in the sera of 1 dog, 8 horses and 19 chickens and to a very slight degree in the sera of 2 humans and 2 cows.

## 16395

## Cold Hemagglutinin in Chinese Kala Azar.

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Rose[1] observed high titers of cold hemagglutinin in two cases of kala azar and concluded that such agglutinin developed as a result of the parasitic infection. This observation has been studied in order to determine its possible clinical significance in this disease.

*Method and Material.* Collection of blood specimens and the titration of the cold hemagglutinin content in fresh serums were carried out strictly according to Rose's method.[1] In nearly all of the tests reported in this paper, group O erythrocytes from one of us, Hou, were used. The final dilution of the serum in the last tube showing fine granular clumpings was recorded as the titer of each test. The serums studied included specimens from 68 proven kala azar patients, 62 normal individuals, and 12 atypical pneumonia cases.

*Results.* The results of the above mentioned investigations are summarized in Table I.

a. *Normal controls.* Cold hemagglutinin with titers varying from 1:4 to 1:32 was present in 56.4% of 62 normal individuals, but absent in the remaining 43.6%. The highest titer recorded in our normal controls was 1:32. Therefore in the present study only titers above 1:32 have been considered significant.

b. *Primary atypical pneumonia cases.* This

[1] Rose, H. M., PROC. SOC. EXP. BIOL. AND MED., 1945, **58**, 93.

TABLE I.
Distribution of Cold Hemagglutinin Titers in 142 Subjects.

| Clinical group | No. of subjects | No. of tests | Cold hemagglutinin titers | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | Neg. | 1:4 | 1:8 | 1:16 | 1:32 | 1:64 | 1:128 | 1:256 | 1:512 | 1:1024 |
| Normal control | 62 | 66 | 30 | 9 | 14 | 9 | 4 | 0 | 0 | 0 | 0 | 0 |
| Kala azar | 68 | 140 | 54 | 18 | 26 | 17 | 13 | 6 | 5 | 1 | 0 | 0 |
| Atypical pneu. | 12 | 20 | 0 | 0 | 1 | 1 | 0 | 5 | 5 | 6 | 1 | 1 |

series of cases served as positive controls. Cold hemagglutinin with titers above 1:32 was present in all of the 12 cases (100%) so diagnosed. The highest value obtained was 1:1024.

c. *Kala azar cases.* Among 68 patients with kala azar, only 11 cases (16.2%) gave cold hemagglutinin titers above 1:32, while 41 patients (60.3%) showed lower titers indistinguishable from those of normal individuals. The highest titer was 1:256.

*Discussion.* Although cold hemagglutinin can be demonstrated in almost every infectious disease, high titers have been rarely encountered except in a few diseases such as primary atypical pneumonia, infectious mononucleosis, trypanosomiasis, measles, mumps orchitis, and scarlet fever.[2,3,4] It might be expected that kala azar, a protozoal infection, like trypanosomiasis, might give high cold hemagglutinin titers as claimed by Rose.[1] The results of our study, however, failed to confirm this. Furthermore, the results of repeated observations made in the course of illness of 68 kala azar patients, indicate that the cold hemagglutinin titer bears no relationship to the severity, stage, and treatment of the disease. For instance, 12 cases complicated by noma (*Cancrum oris*) showed no particular increase in the hemagglutinin titer. Similarly, another fatal case also failed to give a high titer. Of some interest is the fact that one patient, after complete cure for more than one year, still showed a titer 1:64.

As there is some evidence that cold hemagglutinin is an immune body, closely related to serum globulin,[4] our data in 33 kala azar cases were analyzed to see if there is any correlation between cold hemagglutinin titer and serum globulin content. Results shown in Table II failed to demonstrate any such correlation.

*Summary.* Cold hemagglutinin in titers above 1:32 has been found in only 16.2%

2 Stats, D., and Wassermann, L. R., *Medicine*, 1943, **22**, 363.

3 Young, L. E., *Am. J. Med. Sci.*, 1946, **211**, 23.

4 Spingarn, C. L., and Jones, J. P., *Arch. Int. Med.*, 1945, **76**, 75.

TABLE II.
Correlation Between Cold Hemagglutinin Titer and Serum Globulin Content of 33 Kala Azar Cases.

| Serum globul'n content, g% | Cold hemagglutinin titers | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | Neg. | 1:4 | 1:8 | 1:16 | 1:32 | 1:64 | 1:128 | 1:256 |
| 2.1-3 | | | | 1 | 1 | | | |
| 3.1-4 | 5 | 2 | 1 | 1 | 2 | | | 1 |
| 4.1-5 | 1 | 2 | 5 | | | | | |
| 5.1-6 | 3 | | 3 | | | | 1 | |
| 6.1-7 | 1 | 1 | | | | | | |
| 7.1-8 | | | 1 | | | | 1 | |

among 68 Chinese patients with kala azar. The highest titer obtained was 1:256. The titer bears no relationship to the severity, stage, and treatment of the disease. Our results further suggest that there is no correlation between cold hemagglutinin titer and serum globulin content.

## 16396

## Influence of Repeated Anoxia, Electroshock and Insulin Hypoglycemia on Reactivity of Sympathetico-Adrenal System.*

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Numerous investigations[1] have been published by the senior author indicating that procedures such as electrically or chemically induced convulsions, insulin coma and anoxia which are used in the "shock therapy" of mental disease involve an excitation of the sympathetico-adrenal and vago-insulin systems in which the effect on the former system predominates.[2] Further experiments[3] showed that stimulation of the sympathetic division of the hypothalamus has a profound influence on cortical activity. This suggested the possibility that procedures leading to sympathetic discharge and increased reactivity of sympathetic autonomic centers may alter cortical activity not only during the experimental procedures but also for some time afterwards. Consequently, experiments were performed in which the behavioral changes following repeated insulin coma and similar procedures were studied by means of the conditioned reflex method. These studies showed that repeated insulin comas or repeated convulsions led to the restitution of previously inhibited conditioned reactions.[4] This result was interpreted to mean that increased discharges originating in the hypothalamus persisted for some time after the animals had been subjected to coma and convulsions and that these discharges altered the reactivity of those cortical centers which were involved in conditioned reflexes.

If this interpretation is correct, it should be possible to relate this effect to the persistence of increased autonomic reactivity.

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1 Gellhorn, E., *Arch. Neurol. and Psychiat.*, 1938, **40**, 125.

2 For a summary of our earlier work, *cf.* Gellhorn, E., *Autonomic Regulations*, New York, 1943.

3 Murphy, J. P., and Gellhorn, E., *J. Neurophysiol.*, 1945, **8**, 341, 431.

4 Gellhorn, E., and Minatoya, H. J., *J. Neurophysiol.*, 1943, **6**, 161; Kessler, M., and Gellhorn, E., *Am. J. Psychiat.*, 1943, **99**, 687; Gellhorn, E., PROC. SOC. EXP. BIOL. AND MED., 1945, **59**, 155; *Arch. Neurol. and Psychiat.*, 1946, **56**, 216; PROC. SOC. EXP. BIOL. AND MED., 1947, **64**, 375; *Arch. Neurol. and Psychiat.*, 1948, **40**, 125.