was not encountered in those specimens subjected to the longest periods of anoxia. The sites of damage varied widely from experiment to experiment.

Comparison of the present results with those in adult animals[5,6] reveals points of difference which are attributable to characteristics of the young animal. The nerve cells of the newly born appear to be much less vulnerable than those of the adult and withstand anoxia for greater lengths of time. There seems to be less specificity in respect to anoxial damage in the newborn than in the adult.[7]

[5] Weinberger, L. H., Gibbon, M. H., and Gibbon, J. H., Jr., *Arch. Neur. and Psychiat.*, 1940, **43**, 961.

[6] Thorner, M. W., and Levy, F. H., *J. A. M. A.*, 1940, **115**, 1595.

[7] Kabat, H., and Grenell, R. S., *Anat. Rec.*, 1942, **82**, Sup. p. 33.

Summary. Anoxia induced at birth invariably produced symptoms of neural damage. Transient shock, tremors, ataxia, and incoördination were not usually associated with impaired behavior or brain pathology. More than 8 min of anoxia lead to such symptoms as decerebrate states, marked ataxia, convulsions, paralysis, hyper- and hypoesthesia and somnolence, correlated in some cases with behavioral changes. Highly variable nonspecific devastation of brain and cord tissues, occasional hemorrhages, gliosis and generalized atrophy were encountered.

All experiments were controlled with healthy litter-mates and permit the conclusion that neonatal asphyxia (asphyxia pallida) may induce irreparable fortuitous destruction of large or small regions of the brain.

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Response to Murine Poliomyelitis Virus (Lansing Strain) of Mice on Different Levels of Thiamin Intake.*

CLAIRE FOSTER, JAMES H. JONES, WERNER HENLE AND FRIEDA DORFMAN.

From the Departments of Pediatrics and Physiological Chemistry, School of Medicine, University of Pennsylvania, and the Children's Hospital of Philadelphia.

The following is a preliminary report on the relation of thiamin intake to the response of mice inoculated with the Lansing strain of murine poliomyelitis virus.[1] This investigation is part of a long-range program for the study of the relation of diet to resistance to infection.

In each of the 4 experiments summarized here, litters varying from 21 to 31 days of age were split among the groups being compared. Each mouse (except one uninjected group in Experiment I) was injected intracerebrally with 0.03 ml of a 0.5% suspension of mouse brain in saline. Normal mouse brain was used for the controls. Mouse brain infected with the Lansing strain of mouse-adapted poliomyelitis virus served as the virus inoculum. This amount of virus corresponded to between 10 and 100 fifty-percent-mortality doses.

Previous experience had shown that 100 μg of thiamin per 100 g of diet (called diet 100) cover the needs of the growing mouse and that 10 μg per 100 g of diet (called diet 10) lead to signs of deficiency within 15 days.

The first 3 experiments, totaling 646 mice, failed to show any significant difference between mortality of groups on diet 100 inoculated with virus and those on diet 10 inoculated with virus, injected with normal brain, or uninjected. The incidence of paralysis, on the other hand, was markedly greater in the high-thiamin than in the low-thiamin groups inoculated with virus.

Averaging the 3 experiments (each of

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[1] Armstrong, C., *Pub. Health Rep.*, 1939, **54**, 2302.

which was carried 21 days after inoculation), mice which were placed on diet 10 for 15 to 20 days before inoculation with the virus and were continued on it, showed 13% paralysis compared with 74% in the mice on diet 100 for the same period of time. No paralysis was observed in mice injected with normal brain.

The fourth experiment was designed to determine whether or not the thiamin-deficient mice would develop paralysis if the majority of them were prevented from dying of deficiency for at least 21 days after inoculation by increasing the thiamin to a maintenance level.

When the majority of mice on diet 10 became markedly deficient and a few of the smallest ones had died (after 26 days on diet 10), the thiamin in the diet of mice in Groups 2 and 3 (Fig. I and II) was raised from 10 to 30 μg per 100 g of diet. Thereafter, the amount of thiamin given to Groups 2 and 3 was varied in an attempt to keep the majority of these mice alive and just maintain them at approximately constant weight.

All mice were injected 34 days after institution of the experimental diets either with virus (Groups 1 and 2) or with normal brain (Group 3). There was practically no difference in mortality between Group 2 (low-thiamin diet, inoculated with virus) and Group 3 (low-thiamin diet, injected with normal brain). In contrast, the death rate was considerably higher in Group 1 (high-thiamin diet, inoculated with virus). Still more striking was the fact that the incidence of paralysis in mice on the high-thiamin diet (Group 1) was several times that in mice on the diet in which the thiamin was deficient (Group 2). Paralysis was followed by death within a few days. None of the mice injected with normal brain (Group 3) exhibited paralysis.

No explanation is offered at this time for the results observed, nor has the specificity of this reaction yet been investigated. The survival of the virus and the possible histological lesions in the thiamin-deficient mice are being studied in the current experiments.

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