from casein, not considering digestibility. The analysis and caloric value of the stock diet was given in a previous publication.[1] The Maltose diet was supplemented by vitamin feeding daily.[2] The experiment was in progress for 16 days.

Both the control and the experimental rats gained in weight. The former averaged 3.4 gm. per day, the latter 3.2 gm. The average daily intake of food was 14.13 gm. on the stock, and 14.19 gm. on the maltose diet. The daily water intake averaged 20.76 cc. on the stock, and 15.91 cc. on the maltose diet.

The following results were found:

TABLE I.

	Liver Weight gm.	Liver as % of Body Wt.	% of Liver Glycogen	Mgm. Liver Glycogen per Gm. Body Wt.
Stock diet	10.08 ± 0.11	4.37 ± 0.17	4.86 ± 0.14	2.129 ± 0.075
Maltose diet	10.50 ± 0.18	4.50 ± 0.20	5.93 ± 0.24	2.686 ± 0.132

The difference in liver weight is not statistically significant. The liver glycogen formed on the maltose diet is significantly higher than that formed on the stock diet.

If we consider the last 24-hour intake in terms of calories per gram of body weight, we find 0.2785 for the stock, and 0.2854 for the maltose diet. If the liver glycogen, expressed in terms of milligrams per gram of liver, be divided by the caloric intake, the quotient is 175 for the stock and 208 for the maltose rats. The ability to form glycogen is greater on the maltose diet, as would be expected.

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Direct Vestibulo-Spinal Fibers in the Medial Longitudinal Fasciculus of the Cat.*

A. T. RASMUSSEN.

From the Department of Anatomy, Medical School, University of Minnesota.

In spite of a considerable amount of experimental work on the fiber connections of the vestibular nuclei, there are still some points

[1] Greisheimer, Esther M., and Johnson, Olga H., *J. Nutrition*, in press.

[2] Greisheimer, Esther M., and Johnson, Olga H., *Am. J. Physiol.*, in press.

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on which there is disagreement. The marked disturbance in posture and eye movements resulting from vestibular lesions makes it highly desirable to have a very accurate knowledge of the nervous circuits arising from the various vestibular nuclei. To contribute further data on several of the disputed questions, we have produced mechanical lesions in various parts of these nuclei in 20 cats. The cat was used not only because the vestibular system is well developed in this animal, but also because in the course of various other degenerations experiments carried out in this laboratory on the cat, the vestibular nuclei have been unintentionally damaged, thus incidentally contributing data on the vestibular system.

After allowing 2 weeks for degeneration, the brains were kept 3 weeks in 3% potassium dichromate, changing the fluid daily during the first week. Blocks 2 to 3 mm. thick were then kept for 10 days in a relatively large quantity of 3% potassium dichromate containing not over 1/3% osmic acid. Most of the disturbing artifacts are eliminated by thus decreasing the duration in the osmic acid solution and reducing the osmic acid concentration.

In most particulars our series confirm the specific conclusions of Gray[1]: that the superior vestibular nucleus gives origin to fibers which ascend in the medial longitudinal fasciculus of the same side, that the medial (triangular) nucleus sends both ascending and descending fibers into the medial longitudinal fasciculus of the opposite side, and, of course, that the lateral (Deiters') nucleus is the origin of the well known descending lateral vestibulo-spinal tract. In some other particulars, however, we are forced to make somewhat different deductions particularly on the question of direct descending vestibular fibers in the medial longitudinal fasciculus, which Gray was unable to demonstrate.

Extensive unilateral lesions in the region of the medial portion of the vestibular nuclei may result in much descending degeneration in the medial longitudinal fasciculus of both sides; but it appears from the work of others that much of this is due to destruction of cells in the reticular formation outside of the limits of the vestibular nuclei. However, we have succeeded in placing a small lesion in the lower portion of the medial vestibular nucleus (where it is prolonged downwards as the descending nucleus) with slight involvement of the lower pole of the lateral vestibular nucleus. The lesion was made by passing a hypodermic needle, containing a concealed knife, through the cerebellum, caudal to the dentate nucleus, entering the vestibular nuclei just below the stria medullaris acoustica. There

[1] Gray, L. P., *J. Comp. Neur.*, 1926, xli, 319.

being no degeneration in these stria nor in the corpus trapezoideum, shows that the cochlear nucleus was not damaged. Our own work, as well as that of others, show that the passage of the needle through the cerebellum could not be responsible for any of the degeneration in the medial longitudinal fasciculus. Even extensive cerebellar injuries produce no degeneration in this tract. Yet with lesions at the point described and slightly lower but well within the limits of the vestibular nuclei and certainly not directly involving the reticular formation, as generally mapped out in atlases, there may be as much or even more descending degeneration in the medial longitudinal fasciculus of the same side as in the one on the opposite side. The direct fibers are situated more dorsally and laterally than the crossed fibers in the cross-section area of these fasciculi. These fibers extend well into the spinal cord. Since there is no descending degeneration in the medial longitudinal fasciculus after lesions strictly limited to the lateral vestibular nucleus, we conclude that the medial vestibular nucleus or its continuation, the descending nucleus, give origin both to crossed and direct descending spinal fibers via the medial longitudinal fasciculus. Lesions too high in the medial nucleus produce crossed degeneration only.

The only possibility of misinterpretation that we can think of is in connection with the blood supply to the adjacent reticular formation, which might have been interfered with sufficiently by the lesion to have caused degeneration of cells outside of the vestibular nuclei, although there is no evidence of such vascular disturbance.

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Sugar in 0.02 cc. Blood by the Method of Folin and Malmros.

J. F. MC CLENDON.

From the Laboratory of Physiological Chemistry, University of Minnesota, Minneapolis.

The method of Folin and Malmros[1] permits of drawing a second sample of the protein-free blood centrifugate in order to duplicate the determination. In the training of assistants, I have taken advantage of this in order to check their technique but in no case was an absolute check obtained. On trying it myself I did not get an

[1] Folin and Malmros, *J. Biol. Chem.*, 1929, lxxxiii, 115.