

tion. Three nitrogenous supplements were given: milk, casein hydrolyzed by enzymes* and by acid.†

Daily balances for 6 periods of 3 days each were accomplished before the patient was discharged. Three periods were supplemented with enzymic casein hydrolysate, 2 with milk, and one with the acid hydrolysate (Table I). Nitrogen retention was initially avid, but with time more normal retentions were observed. Thus the 3 periods on the enzymic hydrolysate (level 0.2 g N per kilo) showed decreasing retentions of $+.080$, $+.051$, and $+.028$ g per kilo body weight daily. The 2 periods with milk gave values of $+.068$ and $+.053$. These are all exceptionally high retention figures and express the intensity of the patient's need. The 2 supplements are apparently equally effective as nitrogen sources.

* Hydrolyzed with fresh pancreas,³ known under the trade name of Amigen.

† This has previously been shown adequate for growth in rats when supplemented with 2% tryptophane.

³ Mueller, A. J., Kemmerer, K. S., Cox, W. M., Jr., and Barnes, S. T., *J. Biol. Chem.*, 1940, **134**, 573.

The effect of the tryptophane-deficient hydrolysate is of principal interest. The first day of supplementation resulted in a very large urinary nitrogen loss—considerably greater even than the nitrogen intake. The volume of urine for the day was double the usual excretion, and for the period was greater than the previous periods by an average of 700 cc daily. This reversal followed 12 days of large positive retention, and since there was no change in fluid intake, must be attributed to the incompleteness of the acid hydrolysate. Severe loss did not continue, however, since on the second and third day of the period a small positive and small negative balance occurred. These data suggest that the body was able to adjust itself to partially use the deficient mixture. The small net loss, for the 3-day period, $-.019$ g per kilo, is considerably less than the loss during complete protein starvation (Period 1), and confirms similar observations in rats² that even an incomplete source of nitrogen is preferable to no nitrogen at all.

Summary. An enzymic digest of casein may be as effective as milk protein in promoting nitrogen retention, while an acid hydrolysate deficient in tryptophane will not effect nitrogen equilibrium.

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Effects of Lesions of the Periaqueductal Gray Matter in the Cat*

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The great epidemic of lethargic encephalitis directed the attention of neurologists to the region of the midbrain and hypothalamus. A long series of experimental and pathological studies has demonstrated the importance of the gray masses which surround the inferior and posterior parts of the third ventricle, but the posterior extension of the juxta-ventricular gray matter, which sur-

rounds the aqueduct of Sylvius in a cylinder of considerable size, has been neglected. This periaqueductal gray matter is of such a shape, and so located, as to make its destruction very difficult by the usual means—such as the Horsley-Clarke machine—without such extensive damage to neighboring structures as to obscure the interpretation of results.

After much experimentation we developed an electrode which could be inserted by an opening in the occipitoatlantoid ligament

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through the fourth ventricle into the aqueduct and, in this manner, made electrolytic lesions confined to the periaqueductal gray matter of the cat. During the process of development of an electrode which would follow the aqueduct we produced numerous lesions, in all parts of the midbrain, which served as controls since in none of them did the symptoms appear which regularly follow destruction of the periaqueductal gray matter. The lesions were made with an electrolytic apparatus, built for us by Mr. Craig Goodwin, which delivers a constant amperage regardless of variations in the resistance. The location of the lesion was, in each case, verified by serial sections stained alternately with the methods of Nissl and Weil.

When the electrode is merely inserted into the aqueduct, without making an electrolytic lesion, the subsequent behavior of the animals is entirely normal. When the electrode jumps the aqueduct and an electrolytic lesion is made outside the periaqueductal gray matter only alterations in motor performance are noted. If the electrode lies too far forward, and there is destruction of the gray matter in the posterior part of the third ventricle, the cats are catatonic. When, however, the electrode lies accurately in the aqueduct, and the lesion is confined to the periaqueductal gray matter, very striking alterations in the behavior of the animals result.

If the lesion is slight the cats, on awakening from the anesthetic, become very wild. They stare vacantly into space, with pupils widely dilated, and spit, snarl, mew and strike as though seeing imaginary menaces. At the same time they pay no attention to

actual objects in their environment. This state may last for a day or so and disappear leaving a cat with apparently normal behavior.

If the lesion is somewhat more destructive the above mentioned behavior is transitory and poorly developed, passing rapidly in a few hours into the state to be described next.

If the lesion is extensive, the cats lie inert, silent and flaccid as a wet rag. They never again show any interest in food, but must be nourished by stomach tube. They have no spontaneous activity. They will sometimes swallow if milk is allowed to run down in the back of the throat. Irritation which would cause immediate outcry from a normal cat provokes only feeble movements of the head or limbs. The pupils react promptly to light.

If the animal survives for a few days it begins to right itself, first the head and then the entire body. Placing reactions are elicited normally. It may even walk slowly about on occasion if stimulated, but has no spontaneous activity, attends to nothing in its environment and never feeds itself.

The behavior of these animals with destructive lesions of the periaqueductal gray matter resembles strikingly the syndrome seen in human patients after lesions in the brain-stem, described by Bailey, Buchanan and Bucy¹ as arrest of consciousness and by Cairns, Oldfield, Pennybacker and Whitteridge² as akinetic mutism.

¹ Bailey, P., Buchanan, D. N., and Bucy, P., *Intracranial Tumors of Infancy and Childhood*, Univ. of Chicago Press, 1939, p. 250.

² Cairns, H., Oldfield, R. C., Pennybacker, J. B., and Whitteridge, D., *Brain*, 1941, **64**, 273.