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Decorticate and Decerebrate Preparations Produced by Insulin Shock.*

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During insulin shock induced therapeutically there is a progressive depression of nervous functions, in a gradient manner, from the highest (cortical) levels to lower subcortical levels (striatum, midbrain, medullary) which is temporary and reversible.[1, 2] It was decided to demonstrate this gradient effect clinically with the production of animal preparations showing permanent, irreversible lesions at progressively lower levels in the nervous system by prolonging the period of hypoglycemia.

Cats, fasted 18 hours, were given 15 to 20 units of insulin per kilo body weight. When clinical signs indicated early medullary decompensation, small amounts of glucose were given intraperitoneally to prevent death and still maintain a marked degree of hypoglycemia. Hypoglycemia was terminated after 12 to 20 hours. Persistent brain damage was observed in 17 animals. Group I consisted of 5 animals with variable degrees of cortical damage. Group 2 totaled 8 animals with cortical plus subcortical injury ("thalamic preparations"?). Group 3 included 4 cats with loss of function at the midbrain level or lower.

The decorticate preparations (Group 1) showed impairment of vision, impairment of placing and hopping reactions (Bard), slight impairment of righting reactions, difficulty in feeding self, impairment of cleaning habits, restlessness and absence of rage reaction when confronted by a dog. They were able to walk and maintained normal body temperature. These preparations were kept alive from 6 to 90 days.

Preparations in Group 2 showed in addition to the above, inability to walk, decerebrate rigidity, increased tonic neck reflexes and mock rage. These animals were also able to maintain body temperature. These signs point to loss of function at the "thalamic level." The

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1 Angyal, L. V., *Z. f. d. ges. Neurol. u. Psychiat.*, 1937, **157**, 35.

2 Frostig, J. P., *Arch. Neurol. and Psychiat.*, 1938, **39**, 219; also table in *Am. J. Psychiat.*, 1939, **96**, 373.

animals lived 19 to 48 hours after the hypoglycemia was terminated.

Animals in Group 3 were not studied as carefully as the above, but are added because they showed inability to maintain body temperature. They remained in a state of coma until death which occurred from 10 to 18 hours after the interruption of the hypoglycemia. Because of the poikilothermia it is assumed that the damage is at the midbrain level or lower.

Finding clinical evidence of damage at a certain level in the nervous system does not also establish the contention of simultaneous injury of the higher centers. This assumption appears warranted at this time from the clinical course of the animals under the experiment. During the induction of hypoglycemia, the gradient depression of level after level of nervous functions is easily followed and on termination of the hypoglycemia the reversal of this order is obtained. Histopathologic studies are in preparation to establish the proof for this contention.

The demonstration of a gradient effect by insulin in the central nervous system may have a very significant bearing on insulin shock treatment. The effects of this therapy are directed towards psychotic symptoms presumably arising in the highest cerebral centers. Therapeutic effects of the same type now obtained may occur with milder but more prolonged degrees of hypoglycemia, still capable of affecting the highest centers but sparing the lower medullary centers. This would remove the present hazards of shock therapy. This will be subjected to clinical verification.

The technic involved in this study may have an advantage over operative transection or the vascular decerebration of Pollack and Davis for studying the different physiologic strata of the central nervous system. It may be that with insulin the cleavage is along more definite physiological lines than is possible with these other "anatomic" methods.

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