Mezey et al. (1) reported that insulin-induced hypoglycemia stimulated ACTH secretion of normal magnitude in rats with two types of brain lesions (hypophyseal stalk transection and medial basal hypothalamic destruction), either of which "removes all central input to the pituitary." Because treatment with propranolol blocked the ACTH response in such lesioned rats the authors concluded that the ACTH response was driven by systemic elevations in catecholamines. We are puzzled by these results and wonder if, indeed, the separation of the influence of the brain from the pituitary was as complete as the authors intended it to be. Hypothalamic lesions that separate the anterior medial basal hypothalamic from the rest of the brain have been reported to inhibit basal activity in the adenocortical system (2) and to abolish (3) or very markedly inhibit (2, 3) the corticosterone (and presumably the ACTH) response to insulin-induced hypoglycemia. In those studies histological verification of the lesion was carefully and stringently performed. Moreover, medial basal hypothalamic ablation inhibited the rise in corticosterone after hypoglycemia as did pharmacological blockade of hypothalamic input to the corticotrope with chlorpromazine-Nembutal-morphine (4). In the report by Mezey et al., initial ACTH levels in lesioned rats were as high as in unlesioned controls, and the response to hypoglycemia was not blunted. There is another report in which rats with hypothalamic lesions were preselected for their capacity to respond to ether stress; in this selected, responsive group of rats, hypoglycemia induced by large doses of insulin caused a rise in plasma ACTH of normal magnitude (5). These results suggest that in the complete absence of central nervous system input to the pituitary there is a minimal response of the adenocortical system to insulin-induced hypoglycemia in rats, whereas in the presence of some input, the response may be normal. Furthermore, when 2-deoxyglucose was used to produce intracellular glucopenia and a resultant activation of the adenocortical and sympatho-adrenomedullary systems, the magnitude of the corticosterone response to this stimulus was not affected by prior splanchic nerve transection, which markedly diminished the noradrenaline and abolished the adrenaline responses (6). We think that this evidence favors the possibility that the lesions made in the study by Mezey et al. did not abolish all communication between brain and pituitary and that the increase in ACTH secretion resulting from insulin-induced hypoglycemia may not be mediated by catecholamines of peripheral origin in the rat.

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REFERENCES AND NOTES

3. M. Karteszi et al., ibid., 111, 335 (1982).