error of the mean) four hours after the start of immobilization in the canvas sling. Hypophysectomy (five dogs) and adrenalectomy (seven dogs) but not adrenal demedulectomy (five dogs) abolished the response. Median eminence lesions blocked the response to this stimulus and to operative trauma. However, in five animals with lesions in the anterodorsal portion of the hypothalamus, the adrenal response to immobilization was abolished, while the response to surgical trauma remained intact. No area was common to the lesions in these five dogs, but taken together, the lesions covered the area between the region of the anterior commissure and the middle of the median eminence. This suggests that they interrupted afferent pathways descending into the hypothalamus from the limbic cortex.

Finally, Dr. Elaine Smulckoff and I had the opportunity to study four male dogs in which the amygdaloid nucleus had been destroyed bilaterally. Because of the current interest in possible endocrine effects of such lesions, I think it is worth mentioning that we found resticular weight and histology, thyroid radioactive iodine uptake and histology, and adrenal histology normal. These dogs showed a normal hypertrophic response of the remaining adrenal after unilateral adrenalectomy, and their cosinopenic response to operative trauma was also normal. On the other hand, their circulating blood cosinophils fell only 22.7 ± 11.1% four hours after immobilization. This figure is significantly different from the mean fall in normal dogs at the 1% level. These data suggest that the neural impulses initiating ACTH secretion in response to immobilization pass from the amygdaloid region through the anterior and dorsal hypothalamus to the median eminence.

The observations also may have broader implications. It is interesting that the behavioral analysis in the amygdaloidectomized dogs indicated an absence of the behavior normally associated with fear. A similar effect has been reported by others. In the canvas sling, certainly part of the emotional response of normal dogs is one of fear. Is it possible, therefore, that amygdaloid lesions somehow inhibit the emotion of fear; hence the effects of fear, including increased adrenocortical activity, are not seen?

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