REVISTA ESPAÑOLA DE FISIOLOGIA, 30, 49-52. 1974

The Effect of Injury to the Cortical Nucleus of the Amygdala on the Oxidative Metabolism of the Hypothalamus in the Male Rat

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(Received January 23, 1974)

O. SCHIAFFINI and L. FERNANDEZ. *The Effect of Injury to the Cortical Nucleus of the Amygdala on the Oxidative Metabolism of the Hypothalamus in the Male Rat.* Rev. esp. Fisiol., 30, 49-52. 1974.

Earlier work has shown that oxygen consumption by the cerebral amygdala is increased by the gonadotrophins, FSH and LH. This was demonstrated both *in vivo* and *in vitro*. In the present study, experiments were made to determine if a small area of the amygdala could somehow influence the oxidative activity of the hypothalamus of the male rat. For that purpose, lesions were made in the cortical nuclei of the amygdala and the metabolic activity of the hypothalamus was measured 30 days later.

The results obtained indicate that the electrolytic lesion of the cortical nuclei of the amygdala decreases oxygen consumption by the hypothalamus. Oxygen consumption of the cerebral cortex, used as a control tissue, was not affected by the lesion.

These results suggest the possibility of a close physiological relationship between the limbic system and the hypothalamus regarding control of gonadotrophins secretion.

The activity of nervous tissues involved in neuroendocrine processes can be measured indirectly by measuring oxygen consumption and the activity of certain enzymes that have a role in cerebral oxidative metabolism (3).

Castration causes an increase of the oxidative activity of the amygdala and this effect can be cancelled by providing adequate substitutive therapy with testosterone propionate. However, if added *in vitro* to slices of amygdaloid tissue, testosterone propionate fails to cancel the effects of castration (6).

The interruption of the «long» feedback causes an increase in the level of gonadotrophins (4) which may account for the marked increase in the oxidative activity of the amygdala that follows gonadectomy.

The purpose of this study has been to determine whether injury of the amygdaloid cortical nucleus has any effect on the oxidative activity of the hypothalamus.

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Materials and Methods

Adults white male rats (Wistar) fed the standard diet of the Institute of Neurobiology and weighing between 150 and 180 g were used. Light and temperature were controlled and kept constant (25° C, 12 h light and 12 h darkness). Food and water were available *ad libitum*.

Using a stereotaxic device, platinum electrodes were inserted at the points indicated by the DE GROOT coordinates (1) and a direct current of 3 mA was applied. In the case of the control group, the electrodes were inserted as per above but the current was applied at the animal's tail instead of the electrodes. Both groups underwent decapitation 35 days after the operation and the hypothalamus and frontal cerebral cortex were removed in order to determine oxygen uptake in accordance with the methodology described elsewhere (7).

The remaining brain mass was embedded in celloiding and 30 u gallocyanine stained slices were prepared in order to establish the site of the lesion (5).

Results

Figure 1 shows that in the cases where the cortical nucleus was injured, the basal portion of the entorhinal cortex was also affected. The experimental data obtained from animals that did not have bilateral injury of the cortical nucleus or that were affected in neighbouring regions by electrolytic destruction, were discarded. In the cases of the animals whose experimental data were considered, the lesions were approximately 0.9 mm deep to 1 mm in diameter.

Table I. *The effect of injury to the cortical nucleus of the amygdala on the oxidative metabolism of the hypothalamus of the male rat.* Mean standard error of the mean. Between parenthesis the number of determinations.

	Hypothalamus (QO_2: μl O_2/mg wet tissue/h)	Frontal cerebral cortex (QO_2: μl O_2/mg wet tissue/h)
Sham	1.56 ± 0.07 (18)	1.44 ± 0.06 (17)
Injured	1.27 ± 0.06 (10)	1.45 ± 0.07 (10)
Statistical Significance	$p < 0.01$	NS *

* NS: Not significant.

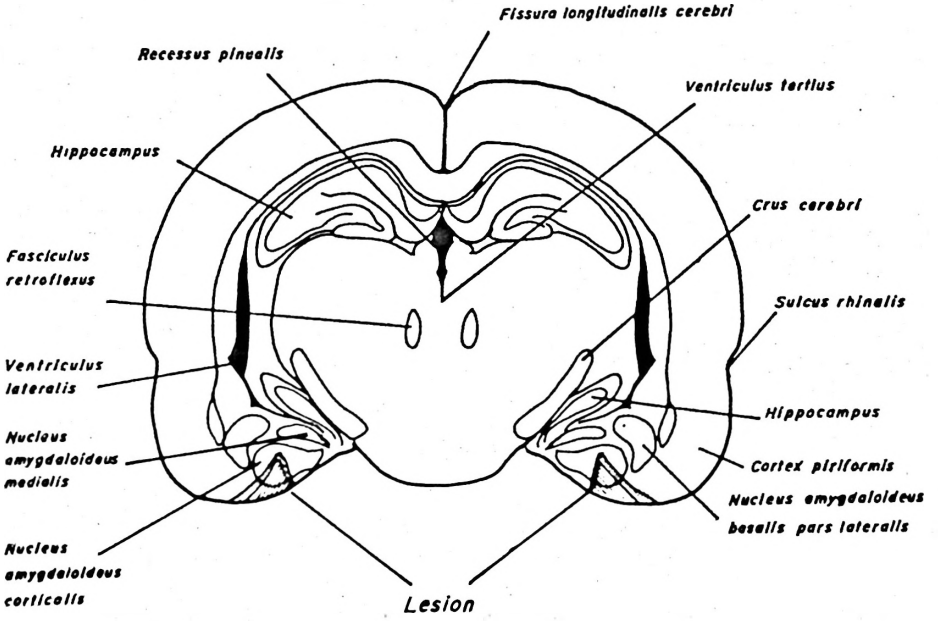

Fig. 1. *Diagram showing the average localization of the lesions.*

Table I shows that, compared to the control group, injury of the cortical nucleus was followed by a profound depression of the oxidative activity of the hypothalamus. On the other hand, no significant alterations were observed in the metabolism of the cerebral cortex. The Student «t» test was used to determine the statistical significance of the differences (2).

Discussion

As demonstrated by *in vitro* experiments where an excess of gonadotrophins is added to amygdaloid slices, the metabolic activity of the amygdala of the male rat is stimulated by LH and FSH. The same conclusion was arrived at from *in vivo* experiments whith hypophysectomized animals (7). On the other hand LH and FSH caused a decrease of the oxidative metabolism of the hypothalamus (3). These findings suggest that the amygdala may be involved in the control mechanism of gonadotrophin secretion.

The finding that a small injury to the amygdaloid region, however indefinite, causes a profound depresion of the metabolic activity of the hypothalamus, would seem to lend support to the view expressed above. Further studies are undoubtedly required before more definite conclusions can be arrived at.

Resumen

En trabajos anteriores, se determinó que las gonadotrofinas FSH y LH ejercían un efecto activador sobre el consumo de oxígeno de la amígdala cerebral. Ello fue demostrado tanto *in vivo* como *in vitro*. En el presente estudio, se trató de observar si una pequeña región de la amígdala podría ejercer alguna influencia sobre la actividad oxidativa del hipotálamo de la rata macho. Con tal motivo se lesionó la región del núcleo cortical amigdalino y se midió, luego de 30 días, la actividad metabólica del hipotálamo.

Los resultados indicaron que la lesión electrolítica del núcleo cortical amigdalino es capaz de reprimir el consumo de oxígeno hipotalámico. La corteza cerebral, de la región frontal, fue utilizada como tejido control y no es afectada por la lesión en amígdala. De lo expuesto, se infiere la posibilidad de una estrecha relación entre sistema límbico e hipotálamo.

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