hypothalamus, without modifications in the middle hypothalamus.

As can be seen in the Table, castration produces a significant decrease in the cytochrome oxidase activity in the anterior and posterior hypothalamic area. The values obtained in the middle hypothalamus were similar in both groups of rats. The administration of testosterone (150 µg twice a week) to castrated rats corrected the modifications observed in the hypothalamic metabolism.

Discussion. In a previous paper it was demonstrated that gonadectomy of male rats produces a decrease in the oxygen uptake of the anterior and posterior hypothalamic area, such modifications being corrected by the administration of testosterone to castrated rats[5]. Studies performed with several substrates of the Krebs cycle showed that succinate and citrate oxidation by anterior and posterior hypothalamus is lower in castrated than in normal rats. Nevertheless, the succinic-dehydrogenase activity of such hypothalamic areas was not modified by gonadectomy[9].

The results of the present paper clearly indicate that castration produces a decrease in the cytochrome oxidase activity in the anterior and posterior hypothalamus and that substitutive therapy restores the values of gonadectomized rats to those found in normal animals.

Cytochrome oxidase activity in different hypothalamic areas

Cytochrome oxidase activity (µl O_2/mg wet tissue h)	Hypothalamus		
	Anterior	Middle	Posterior
(A) Control	1.29 ± 0.09[a] (16)	1.39 ± 0.19 (11)	1.45 ± 0.08 (13)
(B) Castrated	0.86 ± 0.07 (13)	1.36 ± 0.12 (11)	1.04 ± 0.08 (11)
(C) Castrated with testosterone	1.34 ± 0.13 (13)	1.40 ± 0.15 (8)	1.36 ± 0.12 (13)
Analysis of variance			
F ratio	8.33	0.13	4.71
P value	< 0.01	n.s.	< 0.05
Multiple comparisons test			
$P < 0.05$ between:	A *vs* B B *vs* C		A *vs* B B *vs* C

[a] Mean ± standard error. Figures in parenthesis are No. of determinations.

The fact that castration depresses the succinate oxidation by hypothalamus[9] without modifications in the succinic-dehydrogenase activity, and that the cytochroms oxidase of the anterior and posterior hypothalamic areae is less in castrated than in normal rats, seems to indicate that the hypothalamic metabolic alterations produced by gonadectomy are directly related to changes in the respiratory chain (probably between cytochrome C and O_2) of this nervous structure.

Considering that the oxidative metabolism in the central nervous system is one of the principal source of high energy compounds involved in the peptide-synthesizing systems, and remembering the probable nature of the hypothalamic releasing substances[10], it is possible that the modifications in the oxidative metabolism of hypothalamus related with the sexual activity are representative of changes in the synthesis and/or liberation of the hypothalamic releasing factors.

The fact that no modifications were found in the cytochrome oxidase activity of middle hypothalamus in castrated rats is in agreement with previous publications[5,11] in which it has been demonstrated that such hypothalamic area did not modify its metabolic activity during sexual activity.

Resumen. En el presente trabajo se ha estudiado la actividad de la citocromooxidasa en diversas areas hipotalamicas de animales machos castrados y con terapia sustitutiva. Los resultados demonstraron que la castración deprime la actividad de la enzima en el hipotálamo anterior y posterior. La posible implicancia de estos hallazgos con cambios en la cadena respiratoria producidos por la castración es discutida en el trabajo.

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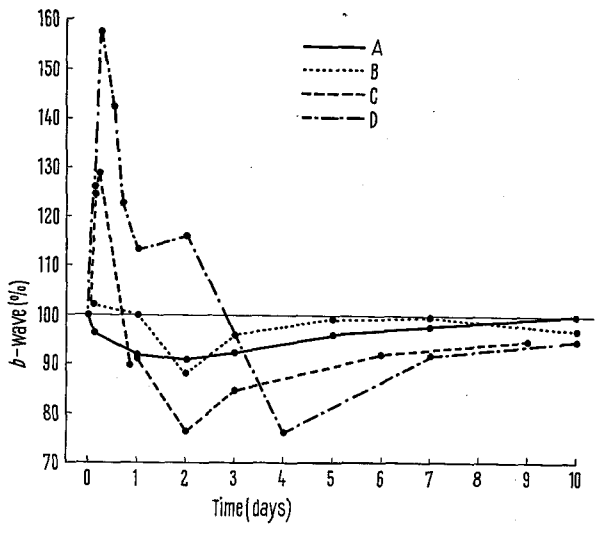
Long-Term Changes in Retinal Function Induced by Short, High Intensity Flashes

For several reasons an increasing interest has been paid during the last decades to the effect on the visual processes of short, high intensity flashes. First, it has been shown that the changes in the rhodopsin cycle following an electronic flash differs from those following longer light exposures[1–6]. Secondly, in light perception experiments, the flashblindness recovery time[7,8] and the development of the foveal after-images[9] following flash exposure indicate that special retinal reactions occur in response to this type of light. Finally, in both experimental and clinical electroretinography there has been an increasing use of short, high intensity electronic flashes. Thus, the early receptor potential[10] and the so-called oscillatory potential[11] are evoked with this type of stimulus. In agreement with the results of the aforementioned light perception experiments, a suppression of the ERG following short, high intensity flashes has been noted[12,13].

In connection with a study on long-term retinal effects caused by different agents, a method has been worked out by which the ERG of the intact rabbit eye can be recorded and evaluated over a period of several months[14].

With this method it was shown that electronic light flashes are followed by reversible long-term changes in the ERG of a type which had not been described before.

The Figure shows the changes in the amplitude of the *b*-wave during 10 days after conditioning flash illumination of the eye. Curve A shows the amplitude changes after a single conditioning light flash and B, C and D the changes following upon a series of repetitive conditioning flashes (conditioning stimulus presented at zero on time axis). The intensity of the test flash was about 2 log units above the *b*-wave threshold of the dark-adapted eye. The experiments illustrated in the Figure were performed on 4 rabbits, each curve representing a series of measurements on 1 animal. In the diagram the amplitude of the *b*-wave is expressed in per cent of the pre-illumination value, which in the 4 experimental series varied between 0.33 and 0.39 mV with a standard deviation(s) for each registration of 2.5%. The intensities of the conditioning light flashes were about 7 log units (curve A and B), 8 log units (curve C) and 9 log units (curve D) above the threshold of the *b*-wave in the dark-adapted eye. When repetitive flashes were given (B, 10 flashes; C and D, 100 flashes) the intervals were 30 sec between flashes. As shown by curve A, even a single light flash with an intensity about 7 log units above the threshold of the dark-adapted eye is followed by a significant reduction of the *b*-wave which lasts for about a week. The conspicuous increase in the *b*-wave, following upon 10 flashes of the same intensity (curve B), is not statistically significant. In order to find out if strong light flashes really can induce an increase of the *b*-wave, an increased flash intensity as well as an increased number of conditioning flashes were used (curve C and D). Curve C shows that 100 flashes with an intensity of about 8 log units above the *b*-wave threshold resulted in a pronounced initial increase of the *b*-wave. When the intensity of the conditioning intermittent light stimulus was raised to about 9 log units above the *b*-wave threshold (D) the initial increase was even larger, lasting about 48 h with a maximum value of 160% 6 h after exposure of the eye to the repetitive conditioning flashes. Following this increase a transient decrease was noted in the *b*-wave and a recovery to pre-illumination values was accomplished in 2 weeks.



Amplitude of *b*-wave (in per cent of pre-illumination value) after illumination with single and repetitive (10 flashes in B; 100 flashes in C and D) light flashes. Intensity of illumination 7 (A and B), 8 (C) and 9 (D) log units above the *b*-wave threshold in the dark-adapted eye.

These data indicate that the decrease in the ERG, as found by BUCKSER[12], may be a reversible process. This latter author noted that the ERG declined over a 12 h period following an electronic flash and he interpreted this as an 'inability' of the ERG to recover.

In connection with the present experiments, the influence of flashes on the *b*-wave threshold of the dark-adapted eye was also studied in the same 4 animals. A reversible increase in the threshold, corresponding to the decrease in amplitude in the Figure, was noted in the A and B series. When the intensity and number of flashes were raised, as in C and D, the increase was preceded by a decrease in threshold, corresponding to the initial increase in amplitude.

This method[14] allows both rabbit eyes to the stimulated simultaneously with light of equal intensity. In an additional experiment an intracranial transection of the left optic nerve was performed. After transection, the flash effects described above were seen in both eyes, although the amplitude of the *b*-wave elicited by the test flash was about 10% higher on the left side than on the right side. It should also be added that similar flash effects were obtained on albino rabbits.

Thus the effects described are not due to influences on a shielding pigment or an efferent mechanism. Further investigations are in progress to determine whether the effect is due to changes in the function of the receptor cells and/or the retinal, neuronal network[15].

Zusammenfassung. Die Versuchstechnik wurde so gewählt, dass die Retinafunktion über längere Zeit registriert werden konnte. Ein Elektronenblitz mit einer Intensität von ca. 7 Logarithmeneinheiten über dem Schwellenwert der *b*-Welle erzielte eine signifikante, oft eine Woche dauernde Verminderung der *b*-Wellenamplitude des dunkeladaptierten Auges. Dies war auch der Fall mit Elektronenblitzen von grösserer Intensität. Der oft viele Wochen dauernden Verminderung ging aber dann eine initiale Erhöhung der *b*-Wellenamplitude voraus. Entsprechende Veränderungen des Schwellenwerts der *b*-Welle wurden auch beobachtet.

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