activated by ipsilateral movements of the jaw without opening the jaw; only a few units were activated by contralateral movements of the jaw. (2) About 15% of the units were inhibited by opening the jaw. Such units exhibited a high discharge rate when the jaw was closed.

All the studied units showed a very modest adaptation. Rubbing the body feathers did not modify the electrical activity of the trigeminal cells of the posterior commissure which was also unaffected by stimulations of the other trigeminal receptors.

In several experiments some muscles of the jaw were gently isolated under ether anaesthesia before the curarization and then they were tested under stretch. The units which responded to the jaw opening were usually activated by ipsilateral stretch of the *adductor mandibulae externus superficialis* and *retractor anguli oris* muscles[6]. The responses of such muscles to moderate stretches exhibited a very short latency: 2–5 msec.

Electrical stimulation of the masticatory muscles during the activation of the unitary discharge provoked by opening the jaw was accompanied by an inhibition of such discharge, identifying the units as muscle spindle afferents.

Summing up, movements of the jaw and stretching of the jaw muscles provoked short latency sustained responses of the trigeminal cells of the posterior commissure on curarized ducks. Such responses were of the type induced by muscle spindles.

Riassunto. Nell'anitra curarizzata è stata registrata mediante microelettrodi di tungsteno la scarica unitaria di cellule trigeminali ubicate nella commessura posteriore. Questa viene modificata dai movimenti della mandibola e dallo stiramento di singoli muscoli che vi si inseriscono. Dette risposte sono del tipo di quelle indotte dai fusi neuromuscolari.

E. Manni, R. Bortolami, and G. B. Azzena

Istituti di Fisiologia Umana e di Anatomia degli Animali domestici dell'Università di Sassari (Sardegna, Italy), February 6, 1965.

[6] E. Oehmichen, Traité de Zoologie *15*, 124 (1950).

Hepatic Parenchymal Cell and Nuclear Volume in Hypophysectomized Toad (*Bufo melanostictus*)

Deb, Boral, and Sarkar[1] and Deb and Boral[2] have recently measured the hepatic parenchymal cell and nuclear volume in the non-hibernating and hibernating toad respectively. Some hormonal influences over cellular proliferation and mitotic activity have been extensively studied by Leblond and Carriere[3] and Distefano and Diermeier[4]. The effect of hypophysectomy over the hepatic nuclear volume in rats has been shown by Lison and Valeri[5]. In the present investigation the hepatic cellular and nuclear volumes in hypophysectomized toad (*Bufo melanostictus*) have been presented and compared to that already reported in the non-hibernating toad.

Male toads of average weight 50–60 g were collected in the wild during their non-hibernating season. Some of the collected animals were hypophysectomized and were kept in this condition for 8 days in the presence of water only. Complete ablation of hypophysis was examined under the microscope. Incompletely ablated animals were discarded. The hypophysectomized toad could survive no more than 10 days, so the experiments were performed after 8 days.

Volume of cell and nucleus was measured in exactly the same way as described in a previous paper by the same authors[1].

It can be observed from the accompanying Table that the volume of hepatic cell and nucleus was significantly reduced in comparison with the non-hibernating ones, both values being statistically significant at the 0.1% level.

From the results presented in the current experiment, it can be concluded that the cellular and nuclear volumes in the liver of hypophysectomized toads are smaller in comparison with the non-hibernating ones. Since McKellar[6] and Tier and Ravanti[7] have shown that the mitotic

[1] C. Deb, M. C. Boral, and C. Sarkar, Anat. Rec. *148*, 499 (1964).
[2] C. Deb and M. C. Boral, Naturwissenschaften *51*, 543 (1964).
[3] C. P. Leblond and R. Carriere, Endocrinology *56*, 261 (1955).
[4] H. S. Distefano and H. F. Diermeier, Proc. Soc. exp. Biol. Med. *92*, 590 (1956).
[5] L. Lison and V. Valeri, Acta endocr. *20*, 257 (1955).
[6] M. McKellar, Am. J. Anat. *85*, 263 (1949).
[7] H. Tier and K. Ravanti, Exp. Cell Res. *5*, 500 (1953).

Cellular and nuclear volume in hypophysectomized and non-hibernating toad (*Bufo melanostictus*)

	Hypophysectomy (10)	Non-hibernation (10)	Difference of mean ± S.E.[a]	*t*
Cell volume – μ^3	589.64 ± 27.8	1377.24 ± 36.84	787.60 ± 46.1	17[b]
Nuclear volume – μ^3	154.94 ± 8.1	231.08 ± 6.44	76.14 ± 10.3	7.4[b]

Results of cell and nuclear volume of non-hibernating toad are taken from a previous paper by the authors[1]. The cipher in parentheses indicates the number of animals.

[a] Individual mean ± S.E. [b] Highly significant.

activity in the liver of adults is much less than in young rats, only adult toads were used in the present investigation. But LEBLOND and CARRIERE[3] and DISTEFANO and DIERMEIER[4] have observed a strikingly reduced mitotic activity in the epithelium of crypts of Lieberkhun and in liver cells, respectively, as a result of hypophysectomy. So it may be concluded that the normal mitotic activity of these tissues may to some extent be associated with the functional activity of the hypophysis in the body. But it may also be mentioned here that after attaining the static mitotic activity in the adult, it is questionable whether the hypophysis has any role in adult cellular mitotic activity. LISON and VALERI[5] have shown the diminution of hepatic nuclear volume in hypophysectomized male rats. Our observations of decrease of both cellular and nuclear volumes in liver as a result of hypophysectomy are in good agreement with their findings[5]. The hypophysectomized toads were kept for 8 days in the presence of water but without any food. Considering the effect of starvation, another group of normal toads (with hypophysis) was also kept in this condition for 8 days and was experimented upon. No significant change in either cellular or nuclear volume has been observed in comparison with normal non-hibernating season's toads. We cannot definitely say at present if the changes as observed in the present investigation were due to changes in metabolic pattern as a result of hypophysectomy, or to any direct imbalance in hormonal action on the cell[8].

Résumé. On a noté une décroissance significative dans les volumes des cellules hépatiques (et aussi dans leurs volumes nucléaires) du crapaud hypophysectomié par rapport au non-hibernant. Ceci est probablement dû à un changement du modèle-type métabolique ou, peut-être, à l'action directe hormonale déséquilibrée, exercée sur les cellules hépatiques à la suite de l'hypophysectomie.

M. C. BORAL and C. DEB

Department of Physiology, University College of Science, Calcutta (India), April 6, 1965.

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Serum-Protidopoyesis in Neonatally Thymectomized Rats Develops Normally

The importance of thymus in the development of immunological powers is proved by the following data: neonatally thymectomized animals accept heterologous skin homografts[1–3], and normal or neoplastic cell-grafts[4,5]; they are unable to form antibodies owing to antigen introduction[2,6–8].

The absence of antibody formation is likely to be a phenomenon dissociated from the ability to synthesize γ-globulins: in new-born thymectomized animals treated with heterologous serum at the age of 2 months, AZAR et al.[9] noticed a plasmocitic reaction and an increase of γ-globulins in the serum quantitatively comparable with those of a normal animal; on the contrary, there is no formation of antibodies. This fact confirms the remark of JANKOVIC et al.[3] on the normality of the serum proteic picture in a neonatally thymectomized animal. On the contrary, MOWBRAY and JEEJEEBHOY[10] appear to have noticed the disappearance of α-globulins in the serum of dogs thymectomized when grown up. We must say that this remark is in contrast with the data of other authors[9,11,12]. The reported data suggest the interest of a complete study on the serum-protidopoyesis in new-born thymectomized animals.

36 Sprague-Dawley strain rats, thymectomized within the first 24 h after birth, were divided into 4 groups of 9 units. The animals of each group (those of the 1st group on the 5th day after thymectomy; those of the 2nd on the 15th day; those of the 3rd on the 30th day; and those of the 4th on the 60th day) received intraperitoneally an aqueous solution of glycine-^{14}C (4 μc^3/g of weight). Within each group, 3 rats were killed 1 h after the injection; 3 rats 2 h after it; 3 rats 24 h after it. Death was caused under ether anaesthesia by loss of blood from neck veins; necroscopy showed the absence of thymic remains. Just after death a fragment of liver and one of spleen were taken and fixed in 10% formalin. Then strips of bone marrow were prepared.

On blood serum total proteins were determined by a biuret method, and paper electrophoresis was executed in double sample in the same separation chamber (barbital buffer, pH 8.7; 0.1 μ; 2.5 V/cm; 12 h). One of the strips was used for the quantitative study of proteic fractions (staining with amido-black). The second strip, sown with 0.1 cm^3 of serum, was employed to evaluate the radioactivity in each fraction (Geiger-Müller counter); the data obtained were expressed as a percentage of the total radioactivity present. The histological pieces, after paraffin inclusion, served for the preparation of autoradiograms (Ilford G5 emulsion; time of exposure 8–10 days).

[1] J. F. A. P. MILLER, Lancet *1961 ii*, 748.
[2] J. F. A. P. MILLER, Ann. N.Y. Acad. Sci. *99*, 340 (1962).
[3] B. D. JANKOVIC, B. H. WAKSMAN, and B. G. ARNASON, J. exp. Med. *116*, 159 (1962).
[4] D. M. V. PARROTT, Transplant. Bull. *29*, 102 (1962).
[5] C. MARTINEZ, J. KERSEY, B. W. PAPERMASTER, and R. A. GOOD, Proc. Soc. exp. Biol. Med. *109*, 193 (1962).
[6] R. A. GOOD, A. P. DALMASSO, C. MARTINEZ, O. K. ARCHER, S. C. PIERCE, and B. W. PAPERMASTER, J. exp. Med. *116*, 773 (1962).
[7] J. F. A. P. MILLER, A. H. E. MARSHALL, and R. G. WHITE, Adv. Immunol. *2*, 111 (1962).
[8] B. W. PAPERMASTER, A. P. DALMASSO, C. MARTINEZ, and R. A. GOOD, Proc. Soc. exp. Biol. Med. *111*, 41 (1962).
[9] H. A. AZAR, R. W. SNYDER, and J. WILLIAMS, Fed. Proc. Fed. Am. Soc. exp. Biol. *22*, 600 (1963).
[10] J. F. MOWBRAY and H. JEEJEEBHOY, reported by R. M. ZOLLINGER, M. C. LINDEM, R. M. FILLER, J. M. CORSON, and R. E. WILSON, New Engl. J. Med. *270*, 704 (1964).
[11] L. D. MCLEAN, S. J. ZAK, R. L. VARCO, and R. A. GOOD, Surgery *40*, 1010 (1956).
[12] L. D. MCLEAN, S. J. ZAK, R. L. VARCO, and R. A. GOOD, Transplant. Bull. *4*, 21 (1957).