Residual oxytocin activity was assayed on the isolated oestrous rat uterus[5,6], 10 μg/l methysergide (Deseril) being added to the physiological solution, in order to exclude the influence of serotonin. Oxytocic activity was assayed against a solution of synthetic oxytocin (Syntocinon) as reference standard. In addition, mixtures of oxytocin and serum from rabbits immunized with oxytocin-albumin conjugate were compared directly with similar mixtures of oxytocin and serum from control animals. The rat uterus preparation was chosen in preference to the chicken blood pressure method to detect oxytocin, since histamine and catecholamines do not contract the oestrous rat uterus, and the effect of serotonin can be abolished by a suitable antagonist. Plasmakinins may, of course, have interfered to some extent.

In the lysine-vasopressin experiments the residual hormone was assayed from its antidiuretic activity on the diuresis of water-loaded rats in alcohol anaesthesia[7-9]. This test was chosen in preference to the rat blood pressure assay, since the antidiuretic activity of the vasopressins is highly specific. It was assumed that the high dilutions employed would eliminate any possible effects due to other substances, such as plasma-kinins, serotonin, histamine, for in this experimental set-up such substances only influence diuresis in relatively high doses. In all the experiments on vasopressin, residual antidiuretic activity after incubating vasopressin with sera containing specific antibodies to vasopressin-albumin conjugate was compared with the residual activity after incubating vasopressin with serum from control animals.

The data pertaining to oxytocin and lysine-vasopressin are summarized in Tables II and III respectively. The data in Table II suggest – but do not prove – that the antibodies produced by oxytocin-albumin conjugate may inactivate oxytocin itself to some extent, at least in certain conditions. This would not appear to hold for lysine-vasopressin (Table III).

The results reported here show that specific antibodies can be produced to oxytocin-albumin conjugate and lysine-vasopressin-albumin conjugate. The antibodies produced by oxytocin-albumin conjugate appeared to have some inactivating effect on oxytocin itself. Those produced by lysine-vasopressin-albumin conjugate did not inactivate lysine-vasopressin.

Zusammenfassung. Mit einem Oxytocin-Albumin-Konjugat bzw. mit einem Lysin-Vasopressin-Albumin-Konjugat konnten bei Kaninchen spezifische Antikörper hervorgerufen werden. Die gegen Oxytocin-Albumin-Konjugat gerichteten Antikörper scheinen auch einen gewissen inaktivierenden Effekt auf genuines Oxytocin zu haben.

L. P. HOLLÄNDER*, J. FRANZ**, and B. BERDE***

Blood Transfusion Centre of the Swiss Red Cross, Basle, Pharmaceutical Chemical** and Biological and Medical*** Research Division, Sandoz Ltd., Basle (Switzerland),*
February 10, 1966.

[5] *British Pharmacopoeia* (Pharmaceutical Press, London 1963).
[6] P. HOLTON, Br. J. Pharmacol. *3*, 328 (1948).
[7] B. BERDE and A. CERLETTI, Helv. physiol. pharmac. Acta *19*, 135 (1961).
[8] W. A. JEFFERS, M. M. LIVEZEY, and J. H. AUSTIN, Proc. Soc. expl. Biol. Med. *50*, 184 (1942).
[9] W. H. SAWYER, Endocrinology *63*, 694 (1958).

Postnatal Variations of the Dry Mass of Neuronal Nuclei of the Spinal Cord in Rat and Guinea-Pig[1]

The behaviour of the volumes of the nerve cell nuclei of the cerebral cortex and of the spinal cord has been studied in histological sections by SUGITA[2], NGOWYANG[3], and PETERS and FLEXNER[4]. The nuclei of the nerve cells can be obtained free in aqueous medium after homogenization of the tissue (VIOLA and PUCCINELLI[5]). With this method it is possible to distinguish nervous from glial nuclei[6] and to determine their exact shape[7] and dry mass by means of the Baker-Smith interference microscope[5].

Using this technique, a comparative study has been made of the volumes and dry mass variations of the nervous nuclei of the lumbar enlargement of the spinal cord in 0–150-day-old rats and guinea-pigs (Table).

The nuclei of the new-born rat have a dry mass and volumes 37% lower than those of 150-day-old rats. After birth both dry mass and volume increase for up to 20 days. The volume and the dry mass do not increase in a synchronous way. 5 days after birth only the dry mass is increased; as a consequence the total solid concentration increases. 20 days after birth the concentration reaches the value of the adult rat. After 20 days no nuclear varia-

Variations of the dry mass and volumes of the neuronal nuclei of the spinal cord (lumbar enlargement) in the rat and guinea-pig with age. 50 determinations were made for each age

Age (days)	Rat			Guinea-pig		
	Dry mass pg	Volume μ^3	Concentration pg/μ^3	Dry mass pg	Volume μ^3	Concentration pg/μ^3
0	38.0 ± 3.1	407.5 ± 31.1	0.096 ± 0.005	55.5 ± 2.5	658.7 ± 45.7	0.087 ± 0.008
5	41.8 ± 1.5	381.8 ± 19.3	0.127 ± 0.004	51.0 ± 2.1	642.9 ± 44.7	0.084 ± 0.009
10	57.1 ± 2.5	473.6 ± 25.7	0.125 ± 0.004	48.5 ± 5.7	698.8 ± 97.6	0.085 ± 0.006
20	64.0 ± 2.4	727.9 ± 38.5	0.091 ± 0.008	54.0 ± 3.0	699.0 ± 67.5	0.084 ± 0.005
150	59.9 ± 3.2	700.1 ± 58.6	0.090 ± 0.002	68.6 ± 3.3	1013.8 ± 69.8	0.079 ± 0.004

tions have been observed and the histogram (not reported) is indistinguishable from that of the adult animal.

In the guinea-pig, different behaviour was observed. The dry mass and volumes do not show any change from birth up to 20 days of life, whereas an increase of about 22% takes place from 20–150 days, without any change in concentration.

In both animals it is likely that the increase is due to variations in insoluble protein content, as the nuclei were isolated in an aqueous medium. In the rat, the increase in dry mass observed during the first 20 days of life can be considered as a continuation of the prenatal growth. In this context it is worth noting that maturation of the motor behaviour is achieved in the rat 15–20 days after birth, when the nuclei of the spinal cord reach their final stage; on the other hand, the guinea-pig's motor behaviour is fully developed at birth. The late increase in dry mass and volume of the neuronal nuclei must be related to other events.

Riassunto. Sono state studiate per mezzo del microscopio ad interferenza Baker-Smith le variazioni di massa secca e volume dei nuclei dei neuroni del rigonfiamento lombare del midollo spinale nel ratto e nella cavia in rapporto all'età.

M. P. Viola-Magni and E. Puccinelli

Istituto di Patologia Generale dell'Università, Pisa (Italy), December 17, 1965.

[1] This investigation was supported by a grant to this Department from the Consiglio Nazionale delle Ricerche (No. 04/76/4/3482).

[2] N. Sugita, J. comp. Neurol. *29*, 119 (1918).
[3] G. Ngowyang, J. comp. Neurol. *50*, 231 (1930).
[4] V. B. Peters and L. B. Flexner, Am. J. Anat. *86*, 133 (1950).
[5] M. P. Viola and E. Puccinelli, Experientia *21*, 320 (1965).
[6] J. I. Nurnberger and M. W. Gordon, in *Progress in Neurobiology, II, Ultrastructure and Cellular Chemistry of Neural Tissue* (Ed., H. Waelsh; Cassel, New York 1957), p. 100.
[7] C. Pellegrino, R. Tongiani, and M. P. Viola, Expl. Cell Res. *53*, 419 (1964).

Accelerated Synaptic Transmission in Nucleus Ventralis-Postero-Lateralis During Deep Sleep[1]

In unanaesthetized cats with chronically implanted electrodes, the mean amplitude of the somatosensory cortex response evoked by peripheral nerve or medial lemniscus shocks is greater during deep sleep than in light sleep[2]. If this change is due to increased excitability of the afferent neurons, then the time required for an afferent volley to reach the somatosensory cortex might also vary as a function of depth of sleep. To test this hypothesis the 1st (cutaneous nerve), 2nd (medial lemniscus) and 3rd (somesthesic radiation) order neurons were stimulated. Mean initial and peak latencies of postsynaptic activity were determined during light and deep sleep at: (1) the medial lemniscus (measuring synaptic transmission time in nuclei gracilis and cuneatus); (2) the somesthesic radiation (measuring synaptic transmission time in the nucleus ventralis-postero-lateralis (VPL); (3) the somatosensory cortex (measuring the latency of the cortical neurons involved). Experiments were performed on cats prepared according to the methods described elsewhere[2]. The mean latency of each type of response was calculated from random samples drawn from each animal. The significance of the differences between the values obtained during light and deep sleep was evaluated according to the analysis of variance (F test).

Results. With cutaneous stimulation (Figure A): (a) The initial latency of the response evoked in the medial lemniscus of 4 animals averaged 3.26 $\pm$ 0.34 msec during light sleep and 3.19 $\pm$ 0.42 msec during deep sleep; the peak latency averaged 4.29 $\pm$ 0.39 msec during light sleep and 4.36 $\pm$ 0.47 msec during deep sleep; neither initial ($-$ 0.07 msec) nor peak ($+$ 0.07 msec) latency changes observed during deep sleep are significant ($P > 0.05$). (b) The initial latency of the response evoked in the somesthesic radiation of 4 animals averaged 6.16 $\pm$ 0.48 msec during light sleep and 5.76 $\pm$ 0.49 msec during deep sleep; the peak latency averaged 8.93 $\pm$ 1.32 msec during light sleep and 8.67 $\pm$ 1.46 msec during deep sleep; the decrease in latency observed during deep sleep (initial $-$ 0.40 msec, peak $-$ 0.26 msec) is highly significant ($P < 0.01$). (c) The initial latency of the surface-positive wave of the response evoked in the somatosensory cortex of 7 animals averaged 7.85 $\pm$ 0.83 msec during light sleep and 7.55 $\pm$ 0.69 msec during

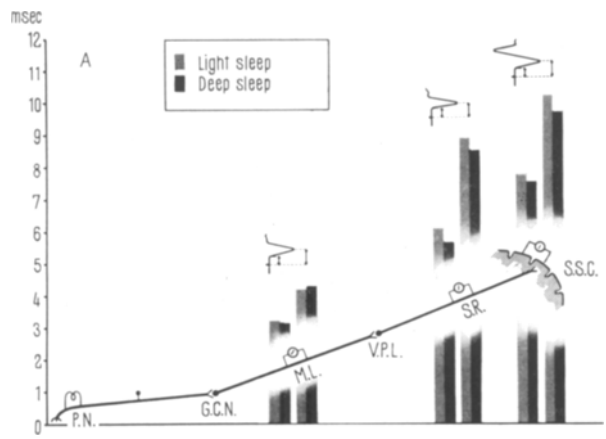

Fig. A. To show latency changes of responses recorded from medial lemniscus (ML), somesthesic radiation (SR) and somatosensory cortex (SSC) upon cutaneous stimulation (PN). Initial and peak latencies are represented under each response by histograms. GCN = gracilis and cuneatus nuclei; VPL = nucleus ventralis-postero-lateralis; msec = milliseconds.

[1] Parts of these results have been presented at a meeting of the Società italiana di Biologia sperimentale, held in Genoa on February 12, 1965.

[2] E. Favale, C. Loeb, M. Manfredi, and G. Sacco, Electroenceph. clin. Neurophysiol. *18*, 354 (1965).