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.

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

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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

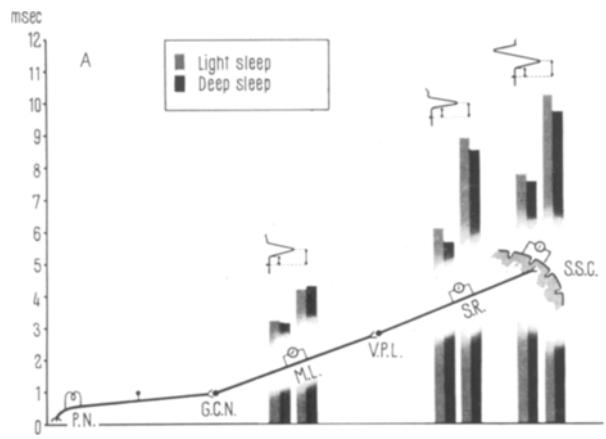

Fig. A. To show latency changes of responses recorded from medial lemniscus (ML), somesthesic radiation (SR) and somatosensory cortex (SSC) upon cutaneous stimulation (PN). Initial and peak latencies are represented under each response by histograms. GCN = gracilis and cuneatus nuclei; VPL = nucleus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

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deep sleep. The peak latency averaged 10.25 ± 1.22 msec during light sleep and 9.82 ± 0.58 msec during deep sleep. Both changes (initial − 0.30 msec and peak − 0.43 msec) observed during deep sleep are highly significant ($P < 0.01$).

With medial lemniscus stimulation (Figure B): (a) The initial latency of the radiation spike (which is thalamic in origin) recorded in the somatosensory cortex of 9 animals averaged 1.39 ± 0.16 msec during light sleep and 1.31 ± 0.13 msec during deep sleep; the peak latency averaged 1.88 ± 0.15 msec during light sleep and 1.78 ± 0.18 msec during deep sleep; the decrease in latency observed during deep sleep (initial − 0.08 msec and peak − 0.10 msec) is highly significant ($P < 0.01$). (b) The peak latency of the surface-positive wave of the response evoked in the somatosensory cortex of 9 animals averaged 4.83 ± 0.66 msec during light sleep and 4.59 ± 0.38 msec during deep sleep. The latency decrease (− 0.24 msec) observed during deep sleep is highly significant ($P < 0.01$).

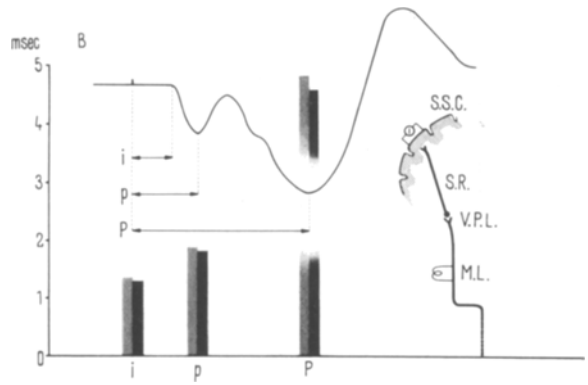

Fig. B. To show latency changes of different components of the response evoked in the somatosensory cortex by medial lemniscus stimulation. Histograms represent initial (i) and peak (p) latencies of the radiation spike; peak latency of the slow surface-positive wave (P). Same symbols as in A.

With somesthesic radiation stimulation the peak latency of the surface-positive wave of the response evoked in the somatosensory cortex of 7 animals averaged 2.18 ± 0.39 msec during light sleep and 2.11 ± 0.27 msec during deep sleep. The latency decrease observed during deep sleep (− 0.07 msec) is not significant ($P > 0.05$).

Conclusion. It is clear that during deep sleep the response evoked in the somatosensory cortex of the cat by shocks to the cutaneous nerve or medial lemniscus is not only higher[2] but has a shorter latency as well. Our experiments demonstrate that this decrease in latency occurs during transmission through the nucleus VPL. No change in synaptic transmission time was observed in nuclei gracilis and cuneatus or in the cortex itself.

Riassunto. La latenza delle risposte evocate nella corteccia somatica sia da stimolazione cutanea che del lemnisco mediale diminuisce significativamente con l'aumentare della profondità del sonno. Tale fenomeno è dovuto ad una accelerata trasmissione degli impulsi ascendenti a livello del nucleo ventro-postero-laterale del talamo.

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The Action of Iodoacetate on the Antigenic Power of Insulin

Confirmed evidence exists that the action of insulin depends, at least in part, on its disulphide group content[1,2] and on the fact that the biological effect of the hormone does not necessarily parallel its antigenic power[3]. This antigenic power of the hormone is, however, inhibited by iodoacetate, although differently from the way this compound inhibits the biological action of insulin in vitro[4-6].

In this work a study is made, using Hales and Randle's immunological method[7], of the action shown by iodoacetate on the antigenic power stability of bovine insulin.

Procedure. A test sample of insulin is treated with iodoacetate, according to the conditions explained in the text, and mixed with a constant amount of insulin ^{131}I. A limited quantity of anti-insulin serum is then added, and the insulin that consequently attaches itself to the antibody is precipitated quantitatively by the incorporation of a second antiserum obtained from the rabbit against the antigenic action of the guinea-pig serum. The precipitated complex is collected by the microfiltration in a millipore membrane and is recounted for radioactivity.

The antigenic power of insulin, as far as its reaction capacity with the specific antibody goes, may thus be assessed by comparison with a standard calculation curve prepared beforehand with pure commercial insulin.

Results. Stability of the antigenic power of insulin in the presence of iodoacetate: Table I shows how a sample of bovine insulin (100 μU/ml), kept at 0–4 °C and at pH 7.4 for 24 h, retains 64 and 85% of its original activity when incubated with $1 \cdot 10^{-4} M$ and $5 \cdot 10^{-5} M$ respectively of iodoacetate.

Action of the incubation time at 37 °C: Table II gives an illustration of the action of different concentrates of iodoacetate when incubated at 37 °C in the presence of a sample of insulin (200 μU/ml) at a pH of 7.4 for 120 min.

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