isolated using the procedure of SIEKEVITZ[13]. Calculated portions of dried protein were plated on stainless steel planchets and counted dry with a thin mica end window G.M. counter. Counting error was less than 2%. Protein was estimated after nitrogen determination with Nessler reagent.

From the data given in the Table it can be seen that leucine incorporation into the microsomal protein by rat liver homogenates is depressed by tridymite ($P < 0.001$): conversely, vitreous silica seems to carry out a less definite action in that sense ($P < 0.05$). It is to be noted that there is a large fluctuation in the experimental data. This is the result of the combined biological and analytical errors, a phenomenon which might also obscure the experimental response if data were incorrectly analysed.

The observations here reported are in keeping with those of ZAMECNIK and KELLER[11] as far as the incorporation rate in control cell-free systems is concerned. The observations show the presence of an inhibitory action by silica dusts on protein biosynthesis at subcellular levels. This action is more pronounced in the presence of crystalline than vitreous silica, an effect which might possibly be related with the well-known less toxic, in vitro, and fibrogenic, in vivo, properties of the latter dust. These observations may actually confirm the derangement of protein biosynthesis[9], a possible specific action of silica in tissues.

Particulate silica in the form of quartz is known to adsorb protein material (e.g. serum proteins and enzymes) and amino acids on its surface[14,15] and to have also oxidative and hydroxylative properties on these latter compounds[16,17]. The decreased protein radioactivity found by the experiments here reported might be the consequence of the peculiar surface activity of particulate silica. As a matter of fact, the extent of the observed inhibition of the amino acid incorporation into the microsomal protein seems to be related to the surface structure of the silica particle tested[18].

Riassunto. È stata studiata l'azione di particelle di silice a struttura cristallina e non cristallina sulla incorporazione di leucina-1-C^{14} nelle proteine microsomiali di fegato.

La silice inibisce l'incorporazione, ma con diversa intensità. Ciò appare essere in rapporto con la struttura delle particelle impiegate.

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Istituto di Patologia Generale dell'Università di Milano (Italy), March 31, 1965.

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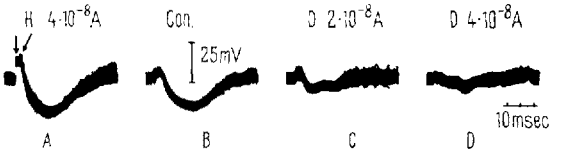
'Disfacilitation' of Red Nucleus Neurones

It has been reported[1] that the large neurones of the red nucleus (RN) receive powerful excitatory postsynaptic potentials (EPSPs) monosynaptically from the contralateral nucleus interpositus (IP) of the cerebellum. On the other hand, stimulation of the cerebellar cortex provokes inhibitory postsynaptic potentials (IPSPs) monosynaptically in the IP neurones, presumably via the corticofugal axons of cerebellar Purkinje cells[2]. Thus it may be expected that the cerebellar cortex acts upon the RN indirectly by inhibiting the IP. This process of disfacilitation, as it may be called, has indeed been demonstrated in the present work.

Cats were used under light nembutal anaesthesia. The experimental procedures were essentially the same as those employed previously[1]. The RN cells were impaled with glass-microclectrodes containing $2M$ NaCl and having electrical resistance of 10–20 $M\Omega$.

In the case of the Figure A–D, the IP region was stimulated with needle electrodes. EPSPs were first induced at latencies of 1.0 and 2.2 msec, respectively (indicated by vertical and oblique arrows), presumably by direct and transsynaptic activation of IP neurones. Thereafter, starting at 6.7 msec a large hyperpolarization occurred, lasting over several ten-milliseconds (Figure B). These observations are in agreement with those by MASSION and ALBE-FESSARD[3,4]. When the membrane potential was displaced by applying hyperpolarizing (Figure A) or depolarizing (Figure C, D) currents, the amplitude of this membrane hyperpolarization was increased or decreased (control in Figure B) in parallel with that of EPSPs. In Figure D the hyperpolarization virtually disappeared together with the EPSPs, indicating that the nul-potential of the hyperpolarization is the same as that of EPSPs. Consequently, the membrane hyperpolarization should not be the IPSP but it should be due to removal of tonically induced EPSPs in RN neurones. Membrane hyperpolarization of the same nature has been seen in ex-



Membrane potential changes produced in a RN neurone by stimulation of IP region. A, during application of hyperpolarizing currents of $4 \cdot 10^{-8}$ A; B, control; C and D, during passage of depolarizing currents of $2 \cdot 10^{-8}$ A and $4 \cdot 10^{-8}$ A, respectively. All records were taken by superposing about 20 faint traces.

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tensor motoneurones during repetitive stimulation of the cerebellar vermis[5,6].

Actually the synaptic noise was found to decrease much during the initial phase of the hyperpolarization. It was also possible to penetrate in the RN region the axons which discharged rhythmically at 30 c/s or so. By the IP stimulation they were first activated with a short latency of 0.3–0.4 msec and thereafter were inhibited for the period of several ten-milliseconds, which corresponded to the initial phase of the hyperpolarization in RN cells. Correspondingly, measurement of the threshold for exciting these axons revealed the prominent excitability decrease in the IP region. It is quite likely that they are axons of the IP cells which discharge tonically and so produce sustained depolarization in RN cells.

When the stimulation was applied to the intermediate part of the anterior lobe of the contralateral cerebellum, similar hyperpolarization could be induced with much shorter latency, its smallest value being 2.2 msec. This latency of 2.2 msec would be accounted for, at least chiefly, by adding 0.7–0.9 msec for inducing IPSP in IP neurones from the cerebellar cortex[2] to 1 msec for evoking EPSPs in RN cells from the IP[1]. The longer latency in Figure A–D, 6.7 msec, could be explained by assuming the IP neurones were inhibited by transsynaptic activation of Purkinje cells through cerebellar afferent fibres as has been seen in Deiter's neurones[7].

Thus the essential feature of the cerebello-rubral system appears to be that the IP mediates tonic facilitatory influences upon the RN under inhibitory control by the cerebellar cortex.

Résumé. Dans les neurones du noyau rouge, il a été demontré par l'enregistrement intracellulaire que les potentiels hyperpolarisants de longue durée sont produits par la stimulation de la région du noyau interposé et du cortex cérébelleux. En changeant le potentiel de membrane de la cellule, l'amplitude de l'hyperpolarisation diminue ou augmente parallèlement avec celle du potentiel postsynaptique excitateur (EPSP). En conséquence, l'hyperpolarisation ne provient pas du potentiel postsynaptique inhibiteur (IPSP), mais d'une réduction de l'effet tonique facilitateur exercé par le noyau interposé sur les neurones du noyau rouge («disfacilitation»).

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Effect of Chlorpromazine on the Testicular Physiology in Rats

The interfering action of chlorpromazine, a tranquilizing agent with adrenal[1] and ovarian[2,3] functions, has been investigated. Several authors ascribe this interference to an action of the drug on the hypothalamus. In this paper, the effect of the same drug on testicular physiology is reported.

Experiments were carried out on a total of 21 adult male albino rats of 130 ± 5 g body weight. The rats were provided with a well balanced vitaminized diet and plain water to drink ad libitum. 14 of the rats were treated subcutaneously with 25 mg/kg chlorpromazine every second day for a 30-day period, and the remaining 7 were taken as the normal controls. At termination of the experiment, all the chlorpromazinized and normal control rats were sacrificed, and testes and seminal vesicles of the respective groups of animals were excised for comparative examinations.

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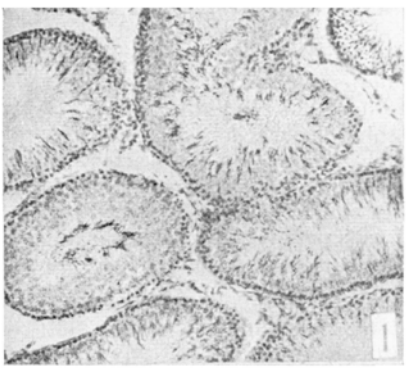

Fig. 1. Showing the histological appearance of testis in normal control rats. × 96.

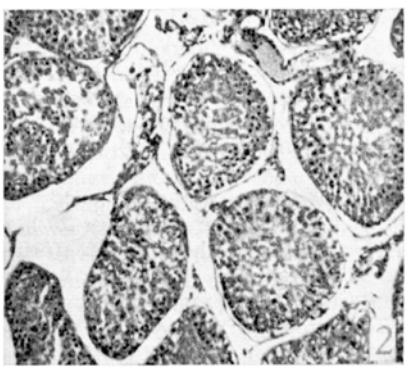

Fig. 2. Testis from the chlorpromazinized rat, showing atrophic changes. Compare with Figure 1. × 96.