was observed throughout most of the explored range of responses (Figures 2B and D). The time course of variability reduction resembled that of the excitability increase of Ia afferent terminals[6]: onset 4–10 msec, peak 30–40 msec and gradual decay up to 100 msec. Variability reduction was also induced by stimulation of the P nerve (with stimulus strengths 1.2–2 T, i.e. times threshold of the most excitable fibres in the nerve).

With stimulus strengths to PL-FDHL smaller than 1.5 T there was facilitation of the MD responses. Onset was about 5 msec, peak at 10 msec and gradual decay up to 30–40 msec after the conditioning stimulus. With greater conditioning stimulus strengths early depression (between 2–5 msec) and facilitation lasting up to 100 msec were frequently observed. In the conditions under which Figures 1 and 2 were obtained, the conditioning volley had a negligible effect on the mean MD responses (Figures 1B, C and 2C). The variance (Figure 2A) and variation coefficient curves (Figure 2D) also remained unaffected.

KATZ and THESLEFF[7] have shown that the input resistance of muscle fibres is inversely related to their diameter. This situation seems to hold also for afferent fibres and motoneurones[8]. Since presynaptic terminals are presumably smaller than motoneurones, one would expect fluctuations of membrane potential to be greater in the former. However, fluctuations of individual elements are only reflected in population responses if their correlation is high[9]. It is therefore suggested that the interneurones ending on the Ia afferent terminals[6] are highly correlated in their spontaneous activities and are, presumably, the main source of variability of the monosynaptic reflex. Reduction of excitability fluctuations of Ia terminals by afferent volleys could be explained: (a) During the PAD produced by the conditioning afferent volleys the conductance of the Ia afferent terminals is probably increased[6]. This would reduce their input resistance and also the membrane potential fluctuations. (b) The long lasting depression that follows activation of the paths leading to PAD[6] would temporarily exclude them as variability sources. Experiments are now in progress to evaluate the 2 possibilities.

Résumé. Sur la préparation de chat spinal aigue, les réponses antidromiques des fibres afférentes Ia dans un noyeau de motoneurones montrent des fluctuations considérables réduites par des volées afférentes qui provoquent une dépolarisation des afférences primaires. Les réponses provoquées par activation directe des motoneurones sont très stables, et leurs fluctuations non affectées par la stimulation afférente. Ces faits suggèrent que la variabilité du réflexe monosynaptique est principalement due aux fluctuations du potentiel de membrane des parties terminales des fibres afférentes Ia.

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Decreased Thyroid Radioiodine Uptake after Diazoxide in Rats

Diazoxide (7-chloro-3-methyl-1, 2, 4-benzothiadiazine, 1, 1-dioxide) is a compound chemically related to thiazide diuretics. The change in the chemical structure, mainly the absence of the free sulfamyl group, results in considerably different biological effects: a high and rapid hypotensive action with a decrease of total peripheral vascular resistance, a reversal of the saluretic action of chlorothiazide in Na-retention and an increased diabetogenic effect. In this paper, the inhibitory influence of diazoxide on the uptake of radioiodine ^{131}I in the thyroid gland of rats is reported.

Method. The radioiodine ^{131}I (in the form of KI) in a dose of 0.2 μCi in 1 ml of physiological saline is injected i.p. to male Wistar rats weighing 220–260 g and fed standard laboratory diet (Larsen). Four h after application of the radioisotope, the animals are sacrificed by coal-gas. Immediately after killing, the thyroid glands are taken out, weighed on the torsion balance, put in the test tubes with 10% NaOH solution and homogenized by boiling. The radioactivity of these preparations is measured in the well-type scintillation counter Tesla. One tenth of the dose of radioiodine is measured likewise as a standard. The results are expressed in % of the dose of the isotope in 1 mg of the thyroid. Five mg of diazoxide (Hyperstat Schering) in the original solution are injected in the tail vein of the rat just prior to the application of the radioiodine. (We are grateful to Schering Comp., Bloomfield, New Jersey for kindly supplying the diazoxide.)

Results. The results are presented in the Table. In the control group of rats the average uptake of radioiodine is 0.65% of the dose in 1 mg of tissue, in 14 rats after the application of diazoxide only 0.49%. The variance of both groups of the values is not statistically different as evaluated by the F test ($p > 0.05$). The comparison of the mean values of both groups by the t test shows a statistically significant difference ($p < 0.01$). In a control experiment, we were unable to prove any decrease of the radioiodine uptake after i.v. injection of 1 ml of physiological

The effect of diazoxide on thyroid radioiodine uptake in rats

Group	No. of rats	^{131}I-uptake %d/mg (mean ± S.E.M.)
Controls	11	0.65 ± 0.04
Diazoxide	14	0.49 ± 0.04 ($p < 0.01$)

saline or of 1 ml of the NaOH solution of pH identical with that of diazoxide solution (pH 11.6). The mean values of the control group (without any injection), after saline and after NaOH were 0.53%/mg, 0.61%/mg and 0.68%/mg respectively.

Discussion and conclusions. FREGLY[1] proved in rats fed with an iodine defficient diet and treated chronically with hydrochlorothiazide, a decrease of the thyroid uptake of radioiodine. This effect is considered to be a probable result of an increased excretion of the iodine. The decrease of the uptake was found in euthyroid patients treated with chlorothiazide[2]. However, the acute administration of hydrochlorothiazide to euthyroid persons did not influence the thyroid radioiodine uptake[3].

In our experiment diazoxide in the administered dose in 1 i.v. injection was able to decrease significantly the thyroid uptake of radioiodine in rats. The mechanism of this effect of diazoxide, which is no diuretic agent, is unknown. It is of interest that diazoxide in the same dose increases the blood flow through the thyroid gland of rats (as indicated by the uptake of radioactive rubidium ^{86}Rb) in the first minutes after the i.v. injection[4]. The elucidation of the presented effects of diazoxide on the thyroid gland and their possible interrelations are a subject of further study.

Zusammenfassung. Es wird über eine signifikante Hemmungswirkung von Diazoxide (Hyperstat Schering) auf die Radiojodspeicherung in der Ratten-Schilddrüse berichtet. Während die Kontrollen einen Mittelwert der Radiojodspeicherung 0,65%/mg zeigen, beträgt er nach i.v. Injektion von 5 mg Diazoxide 0,49%/mg.

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The Early Effects of low DDT Doses on the Nervous System in Animal Experiments

All over the world DDT is one of the most widely used pesticides[1]. Beyond its useful properties, however, it is harmful to fish, warm-blooded animals and even to man[2,3]. In Hungary the amount of DDT accumulated in human fatty tissue is nearly as high as that measured in the U.S.A.[4,5]. KEMÉNY and TARJÁN[6] have demonstrated that even small amounts of DDT might have blastomogenic effects. These findings induced us to elucidate the smallest DDT dose, which, on passing by way of food into the organism, might affect the activity of the nervous system. By demonstrating early symptoms, more serious damage might be prevented. We also wanted to get information about the site of action in the nervous system, about which great discrepancies can be found in the literature[7].

In the present experiments 237 male white Wistar rats weighing 100–130 g each were used. Besides controls, the animals were divided into groups consuming daily, 40, 20, 10, 5, 2.5, 2, 1 and 0.5 mg DDT/kg body weight. Chronic cerebral cortical electrodes were implanted in 77 rats. Then in a state of rest and under loading by means of rhythmic light flashes, EEG recordings were taken at regular intervals from the alert animals moving about unrestrictedly. The curves were evaluated by an analogous electronic computer[8].

For the determination of the site of action before starting the experiment, and on the 3rd, 10th, 17th, 24th and 31st day of DDT administration, 50% of the animals were narcotized with pentobarbital, acting chiefly on the brainstem[9], and the rest with chloralhydrate, acting mainly on the cortex[10], in order to observe which hypnotic would be antagonized by DDT. The time between injection of the narcotic and disappearance of certain position reflexes, as well as the corneal reflex, then the time during which these reflexes could not be elicited, and finally the total time of sleeping were measured in the different groups. The values obtained were evaluated by the 'Gier' electronic digital computer of the Central Statistical Office, performing variation analyses and 'repeated comparison' calculations after SCHEFFÉ[11].

When DDT was applied in an amount of 40 mg/kg body weight, the amplitude, as well as the frequency of the resting EEG activity, increased from the third day. In response to 20 mg the amplitude increased from the third day, and the frequency in the fourth week. In animals treated with 10 mg the frequency did not change, while the amplitude increased measurably from the tenth day above that of the controls. The resting electrical activity of the animals ingesting 5 mg DDT did not change.

When rhythmic flashing-light loading was applied to the rats fed 20, 10 and 5 mg DDT (the group fed 40 mg

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