Resumen. Monos cautivos mantenidos en una dieta vegetariana presentaron niveles séricos de vitamina B_{12} reducidos y lesiones en el sistema nervioso central semejantes a las de la «degeneración combinada sub aguda» del humano. Los nervios periféricos mostraron desmielinización segmentaria, compatible con un compromiso de las células de Schwann al azar. Usualmente hubo remielinización segmentaria, asociada a veces con degeneración axónica (walleriana). No se encontró evidencia de remielinización o de regeneración axónica que pudiera ser atribuída totalmente al tratamiento con vitamina B_{12}.

I. TORRES N., W. TH. SMITH and CH. OXNARD

Departments of Anatomy and Pathology, University of Birmingham (England), 20 October 1968.

Effect of Prostaglandin E_1 on the Strychnine-Induced Convulsion in the Mouse

Prostaglandins were shown to be released from central nervous system and to have some effects on the brain and spinal cord functions[1–5]. In unanesthetized cats, intraventricular injection of prostaglandin E_1 (PGE_1) (7–20 μg/kg) decreased spontaneous activity. However, it reduced spontaneous activity only slightly when given i.v.[1]

In a recent study it was shown that PGE_1 usually caused a delayed long-lasting inhibition of monosynaptic reflex ventral root potentials[6]. PGE_1 was also demonstrated to have a decamethonium-like neuromuscular blocking action in anesthetized cats[7].

In the present investigation the effect of PGE_1 on the convulsion induced by strychnine was studied in mice.

Material and methods. The experiments were carried out on adult, white, male mice from a homogenous strain weighing 20–31 g. The animals were divided into 3 groups of 12 mice each. The first group was injected 2 mg/kg strychnine sulfate i.p. in 0.2 ml saline. This group served as control. The second group was injected 20 μg/kg PGE_1 and the third group 30 μg/kg PGE_1 i.p. (in 0.2 ml saline), 1 min before strychnine administration. The animals were exposed to the same stimuli during the experiments.

The interval between strychnine injection and onset of the first convulsion was recorded. The duration of convulsions and the time of the last convulsion were also recorded. The results were evaluated statistically using Student's *t*-test.

Effect of PGE_1 (20 or 30 μg/kg i.p.) on the convulsions produced by 2 mg/kg i.p. strychnine

	Interval between injection of strychnine and convulsion (min)	Duration of convulsion (min)	Time of last convulsion following strychnine injection (min)
Control (n = 12)	3.25 ± 0.036[a] (3.12–3.45)[b]	1.89 ± 0.077 (1.45–2.16)	5.32 ± 0.042 (5.10–5.50)
After treatment with 20 μg/kg i.p. PGE_1 (n = 12)	5.24 ± 0.034 (5.10–5.50) $p < 0.005$	4.96 ± 0.1 (4.32–5.35) $p < 0.005$	10.34 ± 0.04 (10.11–10.51) $p < 0.005$
After treatment with 30 μg/kg i.p. PGE_1 (n = 12)	7.62 ± 0.07 (7.40–8.05) $p < 0.005$	9.60 ± 0.065 (9.33–10.15) $p < 0.005$	17.51 ± 0.09 (17.16–18.20) $p < 0.005$

[a] Mean ± S.E. of the mean, [b] range, *p*, statistical significance of difference between treated and corresponding controls.

Results. The appearance of the convulsions is significantly delayed in mice treated with 20 μg/kg PGE_1 i.p. In this group, an increase was found in the duration of convulsion and in the time elapse till the last convulsion. The controls and the 20 μg/kg i.p. PGE_1 treated mice did not survive the convulsion. In the 30 μg/kg i.p. PGE_1 treated group, however, all mice survived the convulsion, though the duration of convulsion was found to be longer than in the first and second groups (Table).

Discussion. The antagonistic action of PGE_1 to the lethal effect of strychnine as observed in the third group suggests a dose dependent interaction between the 2 drugs. Several explanations are possible to account for this interaction, including an interference with the absorption and distribution of strychnine. In view of the fact that strychnine increases the level of excitability in spinal neurons by selectively blocking supraspinal inhibition[8], PGE_1 might counteract this disinhibition by strychnine. A similar suggestion was made by DUDA et al.[6] regarding the inhibitory action of PGE_1 on monosynaptic reflexes in cats. This antagonistic effect can also be explained by a neuromuscular action of PGE_1, as observed by KHAIRALLAH et al.[7,9].

Zusammenfassung. Es wurde gezeigt, dass PGE_1 gegen durch Strychnin hervorgerufene Krämpfe deutlich antagonistisch wirkt.

S. DURU[10] and R. K. TÜRKER

Department of Pharmacology, Faculty of Medicine, University of Ankara (Turkey), 6 November 1968.

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[10] Fellow in the Department of Pharmacology, School of Medicine, University of Hacettepe, Ankara, Turkey.