The conduction velocities measured along the geometrical path are typical of small unmyelinated fibres. The geometrical length will certainly be an underestimate of the anatomical path length, but even if the anatomical path is twice or 3 times the length of the geometric path, the conduction velocities are still well within the C-fibre range. The information derived from receptors responding to warming of the rat's scrotum is therefore conducted to the thalamus entirely by a C-fibre pathway.

The fact that skin tissue is translucent at the visible and short IR-wave-lengths produced by the flash[8] and that the receptors will respond to the absorbed radiation has two methodological consequences. First, estimates (such as that in Figure 1) of subcutaneous temperature with opaque sensors must result in an overestimation of the temperature rise following a flash[9]. While any suprathreshold change in temperature will be adequate for the latency determination, if the flash method is used to assess the sensitivity of thermoreceptors[4] then the sensitivity will inevitably be underestimated. Second, although the 'cold' receptors are known to be more superficial than the 'warm' receptors in skin[1] the radiation will penetrate rapidly to both levels. It is therefore not possible to judge whether the central units in the thermosensitive path are responding to suppression of 'cold' receptor activity or enhancement of 'warm' receptor activity. In the particular case of the pathway originating in the rat scrotum, the unimodal distribution of latencies implies that if both 'cold' and 'warm' receptor activity is involved, both types of receptor project centrally through unmyelinated C-fibres.

Zusammenfassung. Mittels photographischem Blitzlicht wurden die Wärmerezeptoren des Ratten-Hodensacks erwärmt und die Nervenleitgeschwindigkeit in der afferenten Bahn bis zum dorsalen Horn und dem ventrobasalen Thalamus gemessen. Bei einer Nervenleitgeschwindigkeit von 0.4 m/sec erwies sich der ganze nervöse Weg als aus marklosen C-Fasern bestehend.

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[8] J. L. Monteith, *Principles of Environmental Physics* (Edward Arnold, London 1972), chapt. 5.
[9] W. B. Nowak, S. Fine, E. Klein, K. Hergenrother and W. P. Hansen, Life Sci. *4*, 1475 (1964).

Glycine, Strychnine and Retinal Inhibition

The evidence that glycine can act as an inhibitory neuro-transmitter was originally presented by Aprison and Werman[1] in 1965. Glycine has been found to have inhibitory actions in the retina in vitro by Ames[2] and in vivo by Korol[3]. There have been no histological or electrophysiological studies of the retina in vivo, which firmly satisfy general criteria for glycine as a synaptic transmitter in the retina. The criteria for evaluation of a substance as a synaptic transmitter is based on its presence, storage, release, postsynaptic action and inactivation.

Cohen[4] showed in mice a large concentration of glycine in the inner retina. The retina uptake of glycine, storage and retention mechanisms were demonstrated by Bruun and Ehinger[5], Ehinger and Falck[6] and Ehinger[7]. The inhibitory action of glycine is antagonized by strychnine[8–12] in other regions of the CNS. Völker[13] showed that strychnine increases the b wave of the rabbit retina in vitro. The previous paper[3] shows the action of glycine on rabbit retina in vivo after intravitreal injection. This was characterized by a reversible loss of the oscillatory potentials of the ERG.

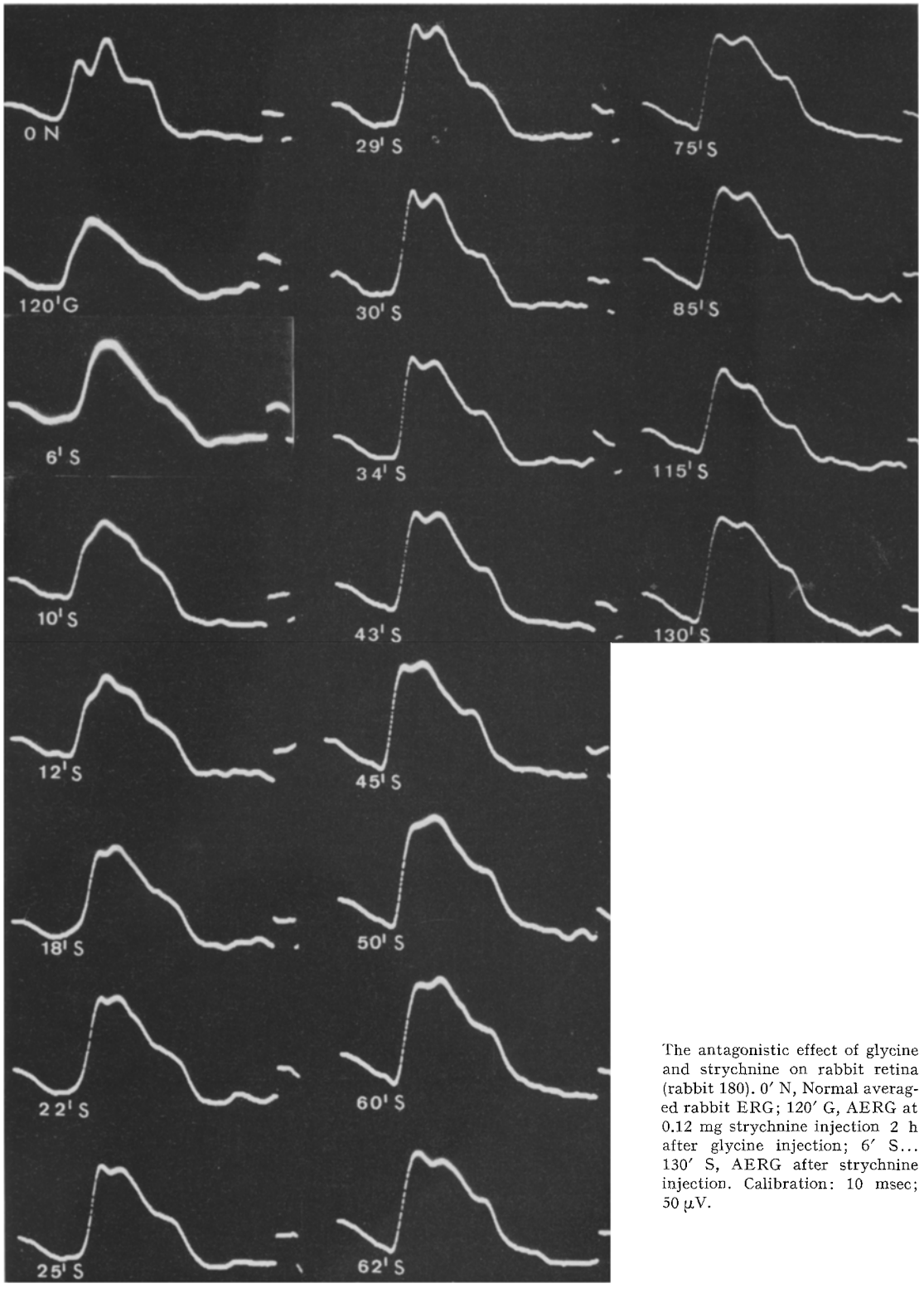
Material and methods. Averaged ERG were recorded on 24 rabbit's eyes (unanesthetised). Group I: 18 rabbit eyes were injected with 3 mg of glycine in the vitreous body to determine time of onset, of maximal effect and of total recovery. Group II: 6 rabbit eyes were injected 2 h after glycine injection with strychnine (2 eyes: 1 mg; 2 eyes 0.25 mg and 2 eyes: 0.12 mg).

Results. Group I: Loss of oscillatory potentials after 1 h of glycine injection. Maximal inhibition between 3 and 10 h with full recovery by 20–24 h. Group II: a) Abolition of b wave of glycine ERG with strychnine 1 mg; b) no effect on b wave of glycine ERG with strychnine 0.25 mg; c) recovery of oscillatory potentials with strychnine 0.12 mg. There was an increase of b wave amplitude 6 min after strychnine and initial recovery of oscillatory potentials 1, 2 and 3, 12 min after strychnine with maximal effect by 45 min (Figure). There was recovery of 3rd OP to normal voltage level and recovery of 1st and 2nd OPs' to an increased voltage level. The effect of strychnine is reversible with recovery of glycine effect by 2 h[14,15].

Conclusion. Glycine has an inhibitory effect on rabbit retina in vivo (group I), evidenced by loss of oscillatory potentials and reduction of amplitude. This effect is reversible and can be antagonized by appropriate concentration of strychnine (group II).

Discussion. Glycine is regarded as putative inhibitory neurotransmitter. The papers of Bruun and Ehinger[5], Ehinger and Falck[6] and Ehinger[7] suggest that glycine may be an inhibitory neuro-transmitter in certain nerve cells of the inner plexiforme layer, mainly the amacrine cells. The present paper shows the inhibitory reversible action of glycine on the rabbit retina observed by ERG's changes. The effect of glycine is antagonized by strychnine

[1] A. Ames and D.A. Pollen, J. Neurophysiol. *32*, 424 (1969).
[2] M.H. Aprison and R. Werman, Life Sci. (Oxford) *4*, 2075 (1965).
[3] A. Bruun and B. Ehinger, Invest. Ophthal. *11*, 191 (1972).
[4] A.I. Cohen, M. McDaniel and H. Orr, Invest. Ophthal. *12*, 686 (1973).
[5] D.R. Curtis, L. Hösli, G.A.R. Johnston and I.H. Johnston, Expl. Brain Res. *5*, 235 (1968).
[6] D.R. Curtis, A.W. Duggan and G.A.R. Johnston, Brain Res. *14*, 759 (1969).
[7] D.R. Curtis, Prog. Brain Res. *31*, 171 (1969).
[8] R.A. Davidoff, M.H. Aprison and R. Werman, Int. J. Neuropharmacol. *8*, 191 (1969).
[9] B. Ehinger and B. Falck, Brain Res. *33*, 17 (1971).
[10] B. Ehinger, Brain Res. *46*, 297 (1972).
[11] L. Hösli and M.J. Neal, Brain Res. *16*, 293 (1969).
[12] S. Korol, Experientia, *29* 984 (1973).
[13] S.E. Smith, J. Neurochem. *14*, 291 (1967).
[14] W. Vörkel and R. Hanitzsch, Experientia *27*, 296 (1971).
[15] R. Werman, R.A. Davidoff and M.H. Aprison, Nature (Lond.) *214*, 681 (1967).



The antagonistic effect of glycine and strychnine on rabbit retina (rabbit 180). 0′ N, Normal averaged rabbit ERG; 120′ G, AERG at 0.12 mg strychnine injection 2 h after glycine injection; 6′ S... 130′ S, AERG after strychnine injection. Calibration: 10 msec; 50 μV.

in appropriate concentrations. The different recovery time for the 1st and 2nd OP in relation with the 3rd OP and amplitude variations suggest a possible different cellular origin or cellular sensitivity to glycine and the antagonistic effect of strychnine.

Résumé. L'injection intravitréenne de glycine (3 mg) chez le lapin entraîne l'abolition des potentiels oscillatoires de l'ERG moyenné photopique, phénomène réversible en 20–24 h. La glycine exerce un effet inhibiteur, qui est annulé par la strychnine en faible dose.

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