intestinal motility is probably brought about by a secondary release of 5-HT.

Secretin had no effect on small intestinal motility. This is in contrast to many recent observations[3] where secretin was often shown to inhibit intestinal motility. Secretin is structurally strikingly similar to glucagon and might therefore have certain similar functional activities. It has been shown previously[4] that the pronounced intestinal inhibition evoked by glucagon is in fact induced by a secondary release of catecholamines from the adrenal medullae. In the present experiments, the catecholamines secretion from the adrenals, known to interfere considerably with intestinal motility, had however, been removed, which might explain the diverging results in this respect.

Gastrin, CCK and secretin caused a significant increase of the regional blood flow. Insofar as gastrin and CCK are concerned, this response was however only transient. After atropine, which abolished the motor response, the blood flow was well sustained and often more pronounced. It appears, therefore, most likely that the tonic contraction on higher concentration might interfere mechanically with the intramural blood flow.

Gastrin, CCK and secretin increased blood flow about 150% at the most. This is a very moderate effect, particularly as compared with the maximal blood flow figures of about 250 ml/min × 100 g tissue recorded when the vascular bed is brought to maximal relaxation by means of supramaximal amounts of isoprenaline. The moderate blood flow increase evoked by the gastrointestinal hormones might be secondary to their metabolic effects on the intestinal glandular tissue, in turn due to the release of vasoactive intermediary substances. This is in accordance with BIBER et al[5], who showed evidence that the 5-HT and α-receptor antagonist dihydroergotamine abolished the blood flow responses to CCK and secretin.

Conclusion. Pentagastrin, CCK and secretin evoke a moderate increase of intestinal blood flow in the cat. Since the tonic contraction interferes mechanically with the blood flow, the increase is only transient following pentagastrin and CCK. After secretin, however, which has no effect on motility, the blood flow increase is sustained.

Zusammenfassung. Nachweis, dass Pentagastrin und CCK, in physiologischen Dosen bei der Katze infundiert, nur geringfügige Durchblutungseffekte im Dünndarmgebiet zeigen, während Sekretin über einen offenbar verschiedenen Mechanismus die Durchblutung erhöht.

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The Effects of Glycine on the Rabbit Retina: Averaged ERG and Averaged Visual Evoked Responses

Evidence exists that glycine can act as an inhibitory neurotransmitter in the central nervous system[1–6]. The retina is the first structure in which the general morphology became known of the neurons putatively operating with glycine as a neurotransmitter. EHINGER and FALCK[7,8] showed in the rabbit retina that the glycine uptake was localized in the neurons and not in the glia. BRUUN and EHINGER[1] showed the characteristics of the glycine uptake into the retinal neurons in in vitro incubation. The uptake mechanism for glycine seems to be specific for retinal neurons. The autoradiographic studies showed that there was a significant accumulation of radioactivity only in certain cells, mainly in the amacrine cells, and diffusely in the inner plexiform layer. Radioactivity was also seen in the ganglion cells, but clearly less than in the amacrine cells. There was very little uptake into other cells, glia included[1].

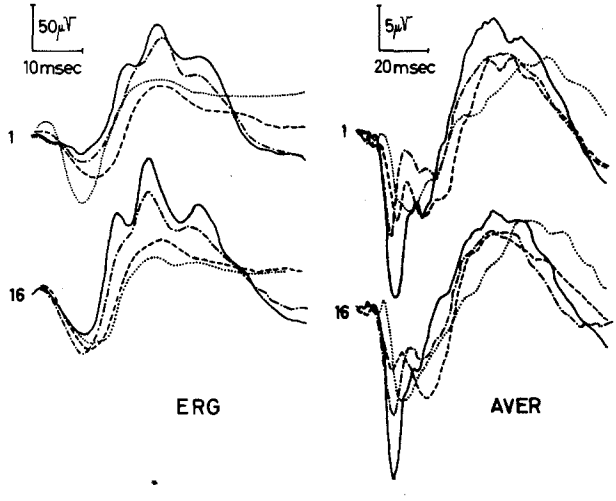

Fig. 1. Simultaneously recorded averaged ERG (left) and averaged VER (right) at different intensities (1: upper, and 16: lower), with a frequency of 1 Hz. Normal ERG and normal AVER. The progressive effect of 0.005 g of glycine after 10 min, 1½ h, and 4 h. Abolition of the ERG's oscillatory potentials and depression of the early components of the the AVER (Rabbit Nr. 70). —, normal; ·-·-·, 10 min; ----, 90 min; ·····, 4 h.

Material and methods. The experiments were carried out on both eyes of six unanesthetised rabbits (i.e. 12 eyes). Averaged photopic ERG and AVER were performed and analysed as previously described (STANGOS[9], KOROL[10]). Glycine 0.005 g was injected into the vitreous body of 10 eyes and 0.002 g into 2 eyes.

The following results have been found (Figure 1 and 2): ERG (Averaged Electroretinogram). 1. A total suppression of the oscillatory potentials (OP) 30 min after the injection of 0.005 g and 90 min after the injection of 0.002 g). 2. The third OP starts to disappear, then the first OP and lastly

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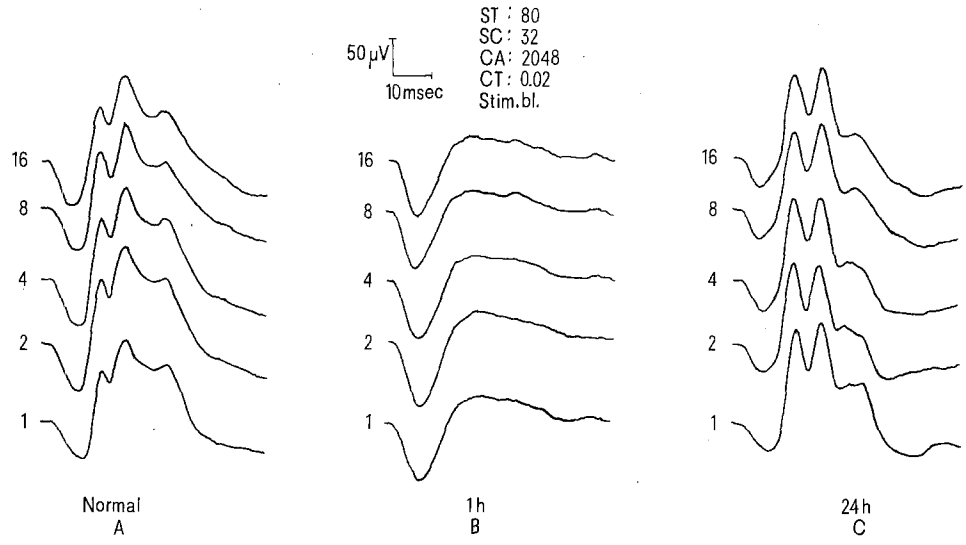

Fig. 2. Averaged ERG at different intensities (1-2-4-8-16) with a frequency of 1 Hz. A) Normal ERG; B) abolition of the oscillatory potentials 1 h after 0.005 g of glycine, and C) recovery of the oscillatory potentials 24 h after glycine (Rabbit Nr. 55).

the second OP disappears. 3. The peaktimes of OP 1 and OP 2 are slightly increased during the decrease. 4. The mean amplitude of the b-wave remains normal, its descending slope is, however, proportionally increased. 5. The averaged latency of the a-wave remains normal but the peak-time and the mean amplitude are increased between 2 to 10 h after the injection of the stronger dose and between 4 to 6 h after the injection of the weaker one. 6. CFF remains normal for the higher intensities of stimulation during the action of the drug but decreases for the lower intensities. 7. Flicker attenuation curves of OP 1, 2 and 3 peak times show a proportionally increased attenuation of the lower frequencies.

AVER (Averaged Visual Evoked Responses). 1. A diminution of the early component with conservation of the later one was found. 2. There was an increased latency of the first negative wave.

Recuperation. ERG. total recovery takes place in 24 h after the injection of the stronger dose and in 10 to 12 h after the injection of the weaker one.

In the first stage of recovery, the first oscillatory potential increases faster than the other two. The peak times of the OP 1 and OP 2 are reduced. The third oscillatory potential showed 2 distinctly separated peaks at 32.5 msec and 42.2 msec. The first of these peaks progressively takes the place of the original OP 3 (X: 35,30 msec $\pm$ 1.89)[9]. The second peak slowly decreases its amplitude and disappears in the descending slope of the b-wave.

AVER. There was a total recovery of the early component in the same time lapse.

Discussion. The origin of the oscillatory potentials has been the subject of many hypotheses. The nature of the oscillations is still obscure[4, 9, 11]. Animal studies, with the aid of intraretinal and intracellular recordings with microelectrodes, combined with electron microscopy studies, further elucidated the origin of the oscillatory potentials. BROWN[10] postulated that the oscillatory potentials are generated in neural feedback circuits in the inner nuclear ayer of cynomolgus monkey's retina. Morphologically a feed-back synaptic arrangement of amacrines and bipolars was suggested by DOWLING[3]. The present work shows the elimination of the oscillatory potentials in the rabbit ERG by glycine with partial blocking of the AVER. Being in accordance with BRUUN and EHINGER[1], our results are quite compatible with the assumption that glycine may be an inhibitory neurotransmitter in certain nerve cells of the inner plexiform layer in the rabbit retina (synaptic amacrine contact ?) with a probable function related to the origin of the oscillatory potential.

Résumé. L'injection intravitréenne de Glycine (0.005 et 0.002 g) chez le lapin entraîne l'abolition des potentiels oscillatoires de l'ERG moyenné photopique, phénomène réversible en 12–24 h. La Glycine exerce probablement un effet inhibiteur sur l'électrogenèse des potentiels oscillatoires.

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Permeability of the Blood-Brain-Barrier in Lymphostatic Encephalopathy Combined with Complex Vitamin B Deficiency. The Protective Effect of Vitamin (Factor) P Treatment

In spite of the well-known absence of lymph vessels from the brain tissue, cervical lymphatic blockage results in an experimental disease, lymphostatic encephalopathy. This syndrome is characterised by various pathophysiological and neuropathological alterations, i.e. by cerebral oedema with an increased permeability of the blood-brain barrier (BBB). This has been demonstrated by means of the electron microscope, using thorotrast as a tracer[1], by the Evans blue fluorescence technique[2] and by chemical analysis of Evans blue uptake by the brain tissue[3]. Recently, SEIDEL and BACK[4], using harmine as an indicator, demonstrated a prolongation of the tremor-inducing effect