Retinoscopy and chromatic aberration

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Summary. The origin of the apparent farsightedness as revealed by retinoscopy in smaller eyes was investigated by using monochromatic retinoscopy on wild rabbits. Our results indicate that a combination of long wavelength light and chromatic aberration of the subject's dioptrics is the major source of this artifact.

Objective measurements of the refractive state of the eye are usually performed by means of retinoscopy. Glickstein and Millodot[2] used this method to measure refraction in several species of mammals. They reported an inverse relation between the measured refractive error and the eye-diameter. They explained this phenomenon[2], which teleologically suggested an artifact inherent in retinoscopy, by assuming that refraction was actually measured with reference to the intersurface between vitreous and retina, whereas in the case of emmetropia an animal's relaxed dioptric system is focused on the layer of receptor outer segments, which in vertebrate retinae is situated at the back of the retina. As the distance between both planes is fairly constant, irrespective of eye-diameter[2], Glickstein and Millodot[2] assume that the error that might be introduced would be inversely related to this diameter.

This explanation of apparent farsightedness takes no account of tapetal and red fundus reflexions that originate from layers just behind the receptor outer segments. In addition it does not take into account the spectral composition of the light used in retinoscopy and the chromatic aberration of the dioptrics of the observed eye. We measured the influence of this chromatic aberration on the results of retinoscopy on rabbits *(Oryctolagus cuniculus).*

No doubt there are several layers within the observed rabbit eye that reflect spectrally more or less neutrally, but clearly the major part of the light that is reflected in retinoscopy is reddish, which indicates that this light is reflected by the choroidal blood vessels just behind the receptor outer segments. An estimate of the luminosity spectrum of this red fundus reflex as seen by a light adapted observer can be made by adding together the logarithmic values of the energy spectrum of the tungsten lamp used, the energy spectrum of the rabbit fundus reflex[3] and the standard photopic visibility function of a human observer. Figure 1 shows the resulting luminosity spectrum peaks in the long wavelength range of our visible spectrum. Actually this type of light is 'infra-red' to the animal[4].

The pupils of 10 wild rabbits were dilated with atropine and 2 observers measured refraction with conventional slit retinoscopy and a series of correcting lenses which escalated in steps of 0.5 diopters. Intermediate values were estimated in steps of 0.1 diopters. The effective ametropia K is given in the table. These data show some discrepancy between the results of the 2 observers. Obviously because of the different criteria used, most of the values obtained by 1 of the observers are somewhat higher than those obtained by the 2nd observer. The general tendency, however, is an apparent hypermetropia of between 1.8 and 2.8 diopters.

From this series of rabbits, we selected 3 specimens -

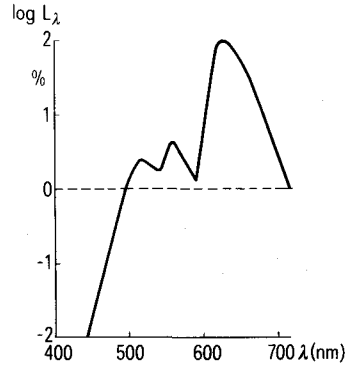

Fig. 1. Estimate of the relative luminosity spectrum (L_λ) of the rabbit red fundus reflex. The logarithmic values of the energy spectrum of this reflex from data of Dodt and Walther[3] are added together with the logarithmic values of both the energy spectrum of the tungsten lamp used in retinoscopy and the human standard photopic visibility function.

Results of refraction measurements obtained by means of conventional slit retinoscopy on 10 wild rabbits. The measurements (n = 10) were performed by 2 observers selected on the basis of minimum variance in results. Obviously as a result of the different criteria that were used, one of the observers tended to give slightly higher values

Rabbit	Effective ametropia in diopters (means ± 95% confidence intervals, n = 10)	
	Observer 1	Observer 2
1	1.7 ± 0.2	1.6 ± 0.1
2	2.0 ± 0.3	1.5 ± 0.1
3	2.2 ± 0.2	1.8 ± 0.1
4	2.4 ± 0.3	1.7 ± 0.1
5	2.4 ± 0.1	1.9 ± 0.1
6	2.2 ± 0.2	2.2 ± 0.1
7	2.1 ± 0.3	2.4 ± 0.1
8	2.6 ± 0.2	2.0 ± 0.1
9	2.4 ± 0.2	2.3 ± 0.2
10	2.9 ± 0.2	2.7 ± 0.1

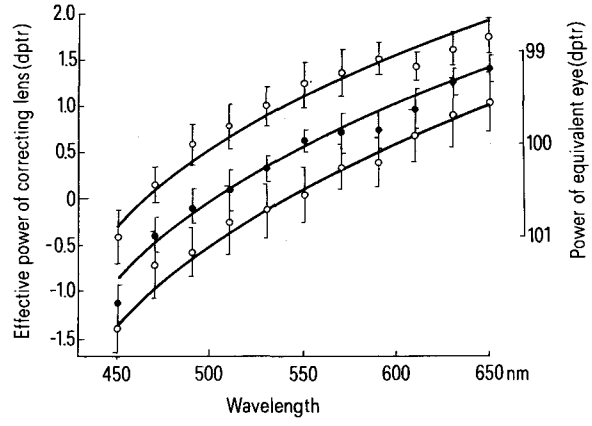

Fig. 2. Results of refraction measurements by means of monochromatic slit retinoscopy on rabbit 1, 3 and 7. Dots: means and 95% confidence intervals of the added lenspower. The results were obtained by 5 observers. Each observation was repeated 3 times on successive days. The curves were calculated following the formula $\Delta F = (\Delta n/n - 1)\ F$, where ΔF is the difference in the refractive power for the interval Δn, while n is the refractive index of water for $\lambda = 500$ nm and F is the total power of an equivalent eye of 100 dptr at 500 nm.

because of their cooperative characters - for monochromatic retinoscopy. With this method the exit slit of a Bausch & Lomb ($\Delta\lambda = 10$ nm) grating monochromator was directed on the subject's eye by means of a beam splitter and a plane mirror that could be rotated around a vertical axis. By rotating this mirror, refraction was measured in the same way as in conventional retinoscopy.

Figure 2 shows for wavelengths between 450 and 650 nm, at 20 nm intervals, the mean and 95% confidence intervals of the added lenspower needed for reaching the 'reversal point' in an experiment in which 5 observers measured refraction.

Clearly the animals were myopic for shorter wavelengths, hypermetropic for longer wavelengths and emmetropic in between. The curves were calculated following the formula $\Delta F = (\Delta n/n - 1)\, F$, where ΔF is the difference in the refractive power for the interval Δn, where n is the refractive index for water for $\lambda = 500$ nm and F is the total power of an equivalent eye of 100 diopters at 500 nm, i.e. the power of the schematic rabbit eye[5].

Our experiments demonstrate that the inherent error in retinoscopy is generally the result of using the wrong wavelength range in these measurements. The range should be adapted to the spectral sensitivity of the subject. In fact retinoscopy has been carried out on many animal species using light that was visible to the observer but 'infra-red' to the animals.

An inverse relation between eye diameter and apparent hypermetropia can therefore be explained on the basis of chromatic aberration. This aberration is linearly related to the refractive power of the dioptrics. This power in its turn is inversely related to the corneo-retinal length, which is assumed to be a relatively constant multiple of the focal length[2].

In human adults the error in retinoscopy will be relatively small because normal eyes are emmetropic for light of 580 nm[6] and because their refractive power is relatively low.

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2 M. Glickstein and M. Millodot, Science *168*, 605 (1970).
3 E. Dodt and J.B. Walther, Pflügers Archiv *266*, 187 (1958).
4 J.F.W. Nuboer and R.H.A. Wessels, Neth. J. Zool. *25*, 398 (1975).
5 A. Hughes, Vision Res. *12*, 123 (1972).
6 R.E. Bedford and G. Wyszecki, J. opt. Soc. Am. *47*, 564 (1957).

Stimulation of gastric secretion by prostaglandin $F_2\alpha$ in rats

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Summary. In rats with chronic gastric fistulas, prostaglandin $F_2\alpha$ stimulated the gastric acid secretion in graded doses of 50, 100, 200 and 400 µg/kg b.wt, while higher doses above 1 mg/kg b.wt tended to inhibit significantly. The gastric antisecretory effect of prostaglandin E_1 could not be altered or modified by subsequent treatment of prostaglandin $F_2\alpha$, while the latter alone without any prior treatment of the former, stimulated output of gastric juice, HCl and pepsin without significantly affecting the concentration of these components.

The inhibitory effect of the prostaglandin E_1, E_2 and A on gastric secretion in the dog and rat have been reported by many[2–4], when administered parenterally or orally. Prostaglandin $F_2\alpha$ has been known to be less potent for inhibiting the gastric secretion[5] in experimental animals, while it transiently inhibited submaximal acid output and increased the frequency of antral contractions in humans when given by continuous i.v. infusion. The effect of prostaglandin on ulcer formation induced by various agents have been studied[6], and the degree of inhibition is considered to be dose-dependent with prostaglandins E_1, E_2 and A, but little is reported on prostaglandin $F_2\alpha$. Although prostaglandin $F_2\alpha$ has been reported to alter the antral motility, gastric blood flow in animals and to inhibit insignificantly basal acid secretion in humans[5], any stimulatory action of it in laboratory rats is not known to us. In the rat, prostaglandin deficiency produces both gastric and intestinal diseases along with the manifestation of ulceration and often stricture[7]. The purpose of the present study was to assess the gastric secretory function following parenteral administration of prostaglandin $F_2\alpha$ in chronically prepared gastric cannulated rats and compare it with other prostaglandins, especially E_1. Rats bearing permanent gastric cannulas were injected i-p. with graded doses of those compounds and the basal gastric secretion collected during 3-h period was analyzed for acid and pepsin.

Material and method. Male Wistar rats, weighing 150–180 g, were implanted with stainless steel gastric cannula under ether anaesthesia following the method of Guha et al.[8]. Gastric secretion in those rats was studied 2 weeks after surgery, following overnight fasting. For collecting gastric juice for a 3-h period during a daily session, the animals were placed in plastic holders. The methods for juice collection and its analysis were previously reported[9]. Experiments were carried out at 2-day intervals, 3 times in a week, at the same time of the day. Initially for first hour, the basal secretion was collected without any treatment which served as their own control, and then at the second hour, prostaglandins $F_2\alpha$ and PGE_1 at different dosage levels, were injected i-p. in identical volumes. The control rats were subjected to the same conditioning procedures except that during second phase, injection of prostaglandin $F_2\alpha$ and E_1 was substituted by saline. The total volume of the collected juice was measured and subsequently analyzed for acidity and pepsin output. Total acid content was determined by titrating with 0.01 N NaOH with phenolphthalein as indicator. Pepsin content was measured following the method of Anson[10] as described earlier (Debnath et al.[11]).

Results. The effect of the i.p. administration of $PGF_2\alpha$ and PGE_1 are summarized in the table. $PGF_2\alpha$ in graded doses produced marked stimulation of gastric secretion as indicated by an increase in the total volume of secretion along with increased acid and pepsin content. The minimum dose of 50 µg/kg was effective in increasing the gastric secretion approximately 50%, while the maximum stimulatory effect was observed at 200 µg/kg. Doses above 400 µg/kg failed to stimulate any further, but higher doses of 1 mg/kg-