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.

1 Acknowledgments. We thank Drs A. van Meeteren and K.W.E.P. Tan for their critical reading of the manuscript.
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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-

Effect of $PGF_{2\alpha}$ and PGE_1 on the gastric secretion of conscious rats

Dose of prostaglandin (µg/kg b.wt, i.p.)	Change in volume of secretion (%)	Change in acid output (%)	Change in peptic activity (%)
$PGF_{2\alpha}$-10	+ 2.16± 1.3	+ 3.4 ± 1.2	+ 5.3 ± 2.3
$PGF_{2\alpha}$-50	+42.36± 6.8	+67.3 ± 8.7	+ 28.2 ± 5.6
$PGF_{2\alpha}$-100	+58.00± 8.6	+73.4 ± 8.5	+ 40.6 ± 8.1
$PGF_{2\alpha}$-200	+65.00± 7.5	+98.6 ± 5.4	+111.3 ±12.3
$PGF_{2\alpha}$-400	+60.00±21.3	+73.7 ± 8.6	+ 72.3 ±15.5
$PGF_{2\alpha}$-1000	+12.2 ± 8.6	+40.3 ± 5.2	+ 48.2 ±12.5
$PGF_{2\alpha}$-2000	−25.5 ±12.4	+35.5 ±15.2	+ 38.5 ±15.5
PGE_1-10	− 2.5 ± 1.5	− 6.5 ± 4.5	− 10.25± 2.5
PGE_1-50	−10.2 ± 3.5	−38.6 ± 3.4	− 28.00± 3.6
PGE_1-100	−29.5 ± 8.6	−58.5 ± 8.6	− 32.5 ± 5.6
PGE_1-200	−58.5 ± 7.8	−71.5 ± 7.8	− 58.5 ± 6.4
PGE_1-400	−52.8 ± 8.2	−30.00± 5.6	− 42.00± 4.5
PGE_1: 200 µg/kg pretreated			
$PGF_{2\alpha}$-50	−42.5 ± 4.5	−38.2 ± 5.5	− 32.00± 3.2
$PGF_{2\alpha}$-100	−62.3 ± 2.3	−59.4 ± 6.5	− 40.5 ± 4.6
$PGF_{2\alpha}$-200	−70.5 ± 3.5	−82.5 ± 5.4	− 68.6 ± 3.2
$PGF_{2\alpha}$-400	−58.6 ± 2.5	−42.8 ± 5.5	− 48.5 ± 3.6

+, Increase; −, decrease; ±, SD, in each dose, total number of rat used = 6.

2 mg/kg produced a depressor response by a marked reduction in the total volume of gastric secretion, although there was marked increase in the total acidity and pepsin concentration. PGE_1 in all doses (50 µg/kg-400 µg/kg) produced marked inhibition of all the gastric parameters. The stimulatory effect of $PGF_{2\alpha}$ at any dose could not be elicited by pretreatment with PGE_1, rather the inhibitory effect of the later was pronounced.

Discussion. The present study demonstrates that $PGF_{2\alpha}$ exhibits a profound and prolonged stimulatory action on gastric acid secretion, while the inhibitory effect of PGE_1 on it is further confirmed in conscious rats with chronic indwelling gastric cannulas[8]. $PGF_{2\alpha}$ at 50 µg/kg dose produced marked increase in total volume of gastric secretion and a dose-dependent response was noted up to 400 µg/kg. Both the acid and pepsin concentration were increased significantly.

Most of the natural as well as synthesized prostaglandins, especially, PGE_1, PGE_2, PGA and methylated analogues of PGE series, have proved to be highly effective depressants of gastric secretion after parenteral administration in experimental animals and man[12]. The mechanism of gastric inhibition by prostaglandins is unknown inspite of several explanations and suggestions on the anti-ulcer activity. Prostaglandins of the E and F series have been reported to exert opposite pharmacological effects in a variety of situations[13,14]. The increased vascular permeability by intradermal PGE_1 administration following bradykinin is inhibited by $PGF_{2\alpha}$[15]. While PGE_1-induced potentiation of the central pharmacological action of morphine[16], phenobarbitone, hexobarbitone and inhibition of pentylenetetrazol convulsions, $PGF_{2\alpha}$ produced a marked inhibition of those actions[17].

In the present study, the stimulating effect of $PGF_{2\alpha}$ was inhibited by PGE_1. PGE_1-induced inhibition of gastric secretion was shown to be serotonin-mediated response; furthermore it itself potentiated the inhibitory effect of serotonin on gastric secretion and intestinal contractility[18]. Further, PGE_1-, PGE_2- and $PGF_{2\alpha}$-induced diarrhoea have been reported to be inhibited by serotonin antagonists[19]. $PGF_{2\alpha}$ has also been reported to reduce rat brain serotonin turnover initially, although later it enhanced the turnover[20]. But PGE_1 has recently been reported to increase the brain and gastric serotonin turnover[21,22]. If it be considered that PGE_1-induced inhibition of gastric secretion is mediated through serotonin by increasing the 5-HT turnover, it may be argued that $PGF_{2\alpha}$-induced stimulation of gastric secretions is perhaps mediated through decreasing 5-HT turnover in the stomach.

The reason why PGE_1-induced prolonged inhibition of gastric secretion was not antagonized or reduced by the stimulatory action of $PGF_{2\alpha}$ is not understood but it is suggested that the stimulatory effect of $PGF_{2\alpha}$ is attributable at least in part to the reduction of 5-HT turnover in the stomach.

1 Acknowledgment. The gift of the prostaglandin E_1 and prostaglandin $F_{2\alpha}$ is acknowledged to Dr J.E. Pike, Upjohn, USA. This paper was read at the IXth Annual Conference of the Indian Pharmacological Society held at Benares (1976) and abstracted in the Indian J. Pharmac. *9*, 73 (1977).

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