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Modification of visual orientation illusions by drugs which influence dopamine and GABA neurones: Differential effects on simultaneous and successive illusions

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Abstract. The effects of haloperidol, nomifensine and lorazepam on the visual tilt illusion were studied in normal volunteers. Haloperidol and nomifensine produced no significant changes in the illusion, although in previous work they had been found to reduce and enhance, respectively, a closely related illusion, the tilt aftereffect. By contrast, lorazepam produced a dose-related increment in the size of the tilt illusion, but had no effect on the tilt aftereffect. The results are discussed in relation to proposed mechanisms which may underlie the two kinds of illusion. The differential effects of individual drugs on the two illusions may reflect their differing actions on two processes: lateral inhibition and adaptation in visual channels.

Key words: Haloperidol – Nomifensine – Lorazepam – Visual tilt illusion – GABA – Dopamine

There is ample evidence that in acute schizophrenia, an illness in which a malfunction of brain dopaminergic mechanisms has been heavily implicated (see Crow et al. 1976 for review) various relatively simple aspects of perception are abnormal. Such evidence comes from the reports of patients themselves (e.g. Chapman 1966; Phillipson and Harris 1985) as well as more objective studies (see, e.g. McGhie 1977 for review). To complement such studies of patients, we have begun to examine the effects of drugs which are known to affect dopaminergic transmission on visual perception in healthy volunteers. Two commonly studied visual effects – the movement and tilt aftereffect (Anstis 1975; Braddick et al. 1978; Frisby 1979) – are perceptual distortions of motion and orientation. The tilt aftereffect (TAE) is the apparent tilt of a line or grating which follows prolonged inspection of a line or grating in a slightly different orientation. The movement aftereffect (MAE) is the apparent motion of a stationary field which follows inspection of a moving field. It has been reported that the MAE is abnormally long in schizophrenia (Abraham and McCallum 1973; Claridge 1967). We have found that the neuroleptic drugs chlorpromazine and haloperidol reduce both the movement and the tilt aftereffects, while the antidepressant nomifensine, which potentiates dopaminergic transmission by preventing the re-uptake of dopamine, enhances the tilt aftereffect (Harris et al. 1983, 1986).

How might the drugs produce these changes? One ex-

planation for the TAE is that it results from the effects of prolonged lateral inhibition in the visual cortex (Tolhurst and Thompson 1975). Thus, during inspection of the adapting stripes, inhibition from the most excited orientation detector is supposed to produce a short-lived reduction in the output of detectors which prefer neighbouring orientations. When the subject subsequently inspects the test stripes, the detectors giving the greatest output may not be those whose preferred orientation corresponds to the physical orientation of the test grating, and so a shift in apparent tilt occurs. So, we speculated, one possible effect of the drugs might be to change the strength of lateral inhibition in the visual cortex. If this speculation is correct, then the simultaneous tilt illusion (TI) should also be affected by appropriate drug treatment. In this illusion (Blakemore et al. 1970), the apparent tilts of lines whose orientation and position are similar are distorted. Figure 1 gives an example, in which the central circular patches of grating are vertical and parallel to each other, but have opposite apparent tilts induced by their surrounding stripes. The distortion is a perceptual expansion of small acute angles. It has been suggested that both this effect and the tilt aftereffect result from lateral inhibition between orientation selective channels in the visual cortex (Tolhurst and Thompson 1975).

It is likely that lateral inhibitory tuning in the visual cortex, where orientation-selective neurons are first encountered in the visual system, is mediated by GABA. This neurotransmitter has a putative inhibitory role in visual cortex (Iversen et al. 1971), and bicuculline, a GABA antagonist, has been shown to reduce or even remove the orientation selectivity of cortical neurones (Sillito 1979). It is plausible to assume that this occurs because bicuculline reduces the inhibitory input to the cell from other orientation-sensitive neurons whose preferred orientation is slightly different.

With these considerations in mind, we have measured the effects of drugs which affect dopaminergic transmission on the TI, and of lorazepam, a benzodiazepine which potentiates the activity of GABA (see Tallman and Gallager 1985 for review), on both the TI and two aftereffects, the TAE and the MAE.

Method

Apparatus and procedure

The tilt illusion. The illusion was measured on a circular area of grating 2.7° in diameter. Immediately surrounding

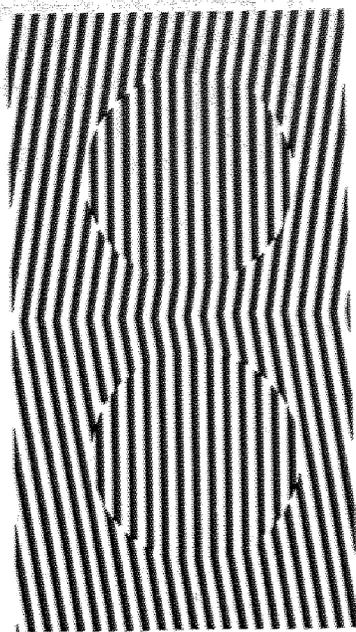


Fig. 1. The Tilt Illusion. The central circular areas of stripes are vertical and parallel to each other, but opposite apparent tilts are induced in them by the surrounding stripes. In the experiments, a single circular area of stripes, surrounded by an annulus of stripes or (for the control condition) a plain field, was adjusted to appear vertical

this test grating was an annulus of inducing grating, inside diameter 2.7° , outside diameter 8.4° . The stripes of the annulus were always tilted 15° clockwise from those of the test grating. This stimulus display was mounted centrally in the right hand half of a grey screen 20° wide by 14° high. It could be rotated by an electric motor controlled by a hand-held remote switch, and its orientation read off from a pointer and scale at the rear of the screen. The right-hand edge of the screen and a thick black stripe mounted on the screen 3.5° from the vertical tangent to the left hand edge of the display provided visual cues to vertical.

The gratings both had a square-wave luminance profile and their spatial frequency was about 9 cycles/degree. The luminance of their white bars was about 48 cd m^{-2} , as measured with an SEI photometer, that of their dark bars about 7 cd m^{-2} . The luminance of the surround was about 20 cd m^{-2} . An annulus of grey card, whose mean luminance was about 30 cd m^{-2} , could be mounted over the annulus of inducing grating. This and the TAE and MAE displays were illuminated by overhead fluorescent room lights, in a room with closed blackout blinds.

The subject sat at 213 cm, and fixated the centre of the test grating. His task was to adjust the grating until it appeared vertical. A block of five readings was taken with the grey masking annulus in position and a block of five with the inducing stripes visible. Each setting was disturbed once the reading had been made.

Tilt aftereffect. The apparatus and procedure have been described elsewhere (Harris et al. 1983). The 4° diameter adapting and test fields were mounted side by side in a black surround. Their spatial frequency, contrast and luminance were the same as for the tilt illusion, at the same viewing distance. A piece of card like the surround covered

one of the gratings. A block of five settings to vertical was made on the test grating with the adapting grating covered. Then two blocks of five adapting trials were run, in which the subject scanned along a horizontal bar in the middle of the adapting grating (with the test grating covered) then adjusted the test grating to appear vertical while gazing at its centre (with the adapting grating covered). Settings were disturbed between trials. In one block, adaptation lasted for 30 s, in the other 40 s. The time taken from the end of adaptation to make each setting was measured with a stop clock.

Movement aftereffect. The apparatus and procedure have been described elsewhere (Harris et al. 1983). A $5^\circ 38 \text{ min}$ diameter rotatable (5 rpm) field of small circular dots (10 min) was back-projected on a Perspex screen. Surrounding this field was a 11° square area of random texture. Viewing distance was 183 cm. Two blocks of five tests were made, one of 30 s, the other of 40 s adaptation. The subject gazed at the centre of the display, the movement was turned on for the appropriate time, then turned off, and the reported duration of the MAE timed with a stop-clock.

Drowsiness and simple reaction time. Subjective estimates of drowsiness were made on analog scales. The data for haloperidol, nomifensine and maprotiline have been reported elsewhere (Harris et al. 1986). For lorazepam, the subject was asked to place a mark corresponding to his present state of alertness on a 16 mm long straight line. One end of the line was designated "hyperalert", the other "barely awake", and the mid-point "normal for time of day". To measure RTs, the subject gazed at a small red LED, and had the press a push-button as soon as possible after the LED was illuminated. A microcomputer measured the interval between LED illumination and response to the nearest millisecond. Again, the data from haloperidol, nomifensine and maprotiline have been reported elsewhere (Harris et al. 1986). For lorazepam, two blocks of ten RT measures, one at the beginning and one at the end of the test session were run.

Drugs

Haloperidol was given by mouth at a dose of 0.05 mg/kg. This results in plasma concentrations of drug in the range 2–4 ng/ml serum (Forsman and Ohman 1974). Since peak plasma concentrations are found 3–6 h after oral dosage, the drug was given at 11.00 hours and testing begun at 14.00 hours. It was compared with inert placebo in 14 normal healthy volunteers (nine male, five female), age range 19–34 years, in a repeated measures design. On their first visit to the laboratory, half the subjects received placebo, half haloperidol. At least 6 clear days were left between successive experimental days, except for three subjects for whom the interval was 3 days, and two subjects for whom it was 2 days.

Oral nomifensine was given at 12.00 hours at a dose of 50 mg, 2 h before testing. This regime has been reported by others to yield peak plasma concentrations in the range 93–177 ng/ml (Brogden et al. 1979).

Nomifensine blocks reuptake both of noradrenaline and dopamine. To control for effects of this drug on noradrenergic uptake sites, parallel tests were done using maprotiline which, at low concentrations, acts highly specifically against

noradrenaline uptake sites in rat brain (Brogden et al. 1979). Maprotiline was given as an oral dose of 50 mg at 09.00 hours, and testing begun at 14.00–16.00 hours, a time when plasma concentrations are rising rapidly. Peak concentrations of this drug are not attained until about 10 h after administration.

Nomifensine and maprotiline were compared with each other and with placebo in 15 normal subjects, (ten male, 18–22 years), in a repeated measures experiment. The order of drug administration was randomised over subjects. At least 6 days occurred between administration of different drugs, except for one subject for whom it was 5 days; for three subjects, 4 days; and for one subject, 2 days.

Lorazepam was given by mouth at 0.015 or 0.030 mg/kg 1 h before testing, at a time when near peak plasma concentrations may be achieved (Dundee et al. 1978). These two doses were compared with each other and with placebo in 12 subjects (all male, 19–24 years), in a repeated measures design. The minimum interval between successive experimental days was at least 5 clear days, except for four subjects, for whom one interval was 2 days.

The subjects were almost all students, mostly of medical sciences, who were paid for their participation, and informed about the broad aims of the study. Perceptual and other tests were done double-blind.

Results

For the TI, each setting made with the masking annulus in position was subtracted from the corresponding setting with the inducing stripes visible. Each pre-adaptation setting on the TAE display was subtracted from the corresponding post-adaptation setting. These five difference scores were treated as measures of the TI and TAE, respectively, and the means calculated for each block. The results from the TAE and MAE for the neuroleptics chlorpromazine and haloperidol, and the antidepressant nomifensine have been reported elsewhere (Harris et al. 1983, 1986). The TI results for haloperidol and the antidepressant are shown in Fig. 2. Haloperidol tended to enhance the illusion, but the effect was not significant: ($t=1.59$; $df=13$; $P>0.05$). The mean tilt illusions for placebo and maprotiline were almost identical, and although nomifensine tended to reduce the illusion compared with maprotiline and placebo, the effects were not significant [from the drug (3) by subject (15) analysis of variance, $F=1.29$; $df=2,28$; $P>0.05$]. These findings contrast with the corresponding results on the tilt aftereffect found in earlier experiments. Thus, haloperidol significantly reduced both tilt and movement aftereffects, while nomifensine significantly enhanced the tilt aftereffect (Harris et al. 1986).

The effects of lorazepam on all three visual tests are shown in Fig. 3. There was a clear enhancement of the TI, with a dose-dependent effect, 0.015 mg/kg giving a larger effect than placebo and smaller than the 0.030 mg/kg dose. From the drug (3) by subject (12) Analysis of Variance this effect was significant ($F=4.40$; $df=2,22$; $P<0.025$). Although the F ratios reflect the consistency of the drug effects, one can ask, for example, how many subjects produced results in the opposite direction to the means. For the tilt illusion, after lorazepam, (which gave a significant enhancement) only 2/12 produced an illusion after the high dose of drug which was smaller than that after placebo. For haloperidol, (which gave no significant enhancement

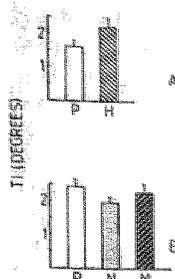


Fig. 2. A, B. Effects of various drugs on the Tilt Illusion. The histogram bars represent means to which each subject contributed five readings, and the vertical bars standard errors. A Comparison of placebo (P) with 0.05 mg/kg haloperidol (H). Number of subjects ($n=14$). B Comparison of placebo (P) with 50 mg oral nomifensine (N) and 50 mg oral maprotiline (M). $n=15$

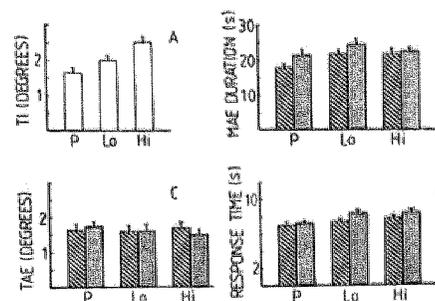


Fig. 3. A–D. Comparison of the effects of placebo (P), 0.015 mg/kg (Lo) and 0.030 mg/kg (Hi) oral lorazepam on three visual effects, and the response times to set the TAE test grating to apparent vertical. In Figs. 3b, c, and d, hatched histogram bars represent data from 30 s adaptation, stippled bars from 40 s adaptation. $n=12$. A The tilt illusion. B The movement aftereffect. C The tilt aftereffect. D Response times to make TAE settings. Other conventions as Fig. 2

of the TI) 6/14 gave a smaller TI after haloperidol than placebo, with one subject giving identical illusions in the two drug conditions. For the anti-depressants (when no significant changes were found), 7/15 subjects gave a larger tilt illusion after nomifensine than after placebo and 7/15 for nomifensine compared with maprotiline.

In contrast to the tilt illusion, lorazepam had little effect on the TAE [from the drug (3) by adapting time (2) by subject (12) ANOVA, for the drug effect, $F=0.12$; $df=2,22$; $P>0.05$; and for the adapting time effect, $F=0.108$; $df=1,11$; $P>0.05$]. The small increase in response times to set the TAE test grating to appear vertical was not significant (from the ANOVA, $F=2.54$; $df=2,22$; $P>0.05$), so that it is unlikely that any drug effect on the TAE was nullified by greater decay of the aftereffect. There was a small enhancement of the MAE by the drug, but this was not significant [from the drug (3) by adapting time (2) by subject (12) ANOVA, $F=2.05$; $df=2,22$; $P>0.05$], and was not related to dosage in the same way as the TI result. A significant effect of adapting time was found for the MAE, the longer adapting time giving significantly longer aftereffects ($F=16.21$; $df=1,11$; $P>0.01$). The consistency of the measurements reflected this lack of significance. Averaged over the two adaptation durations, 6/12 subjects gave a smaller TAE after the high dose of lorazepam than after placebo, and 5/12 a shorter MAE after the high dose of lorazepam than after placebo.

Lorazepam increased subjective drowsiness. (The values given are the mean displacement from the midpoint of the scale, and a minus means a change towards greater drowsiness.) For the placebo condition, the shift (in cm) was -0.57 (sd 1.08), for the low dose -1.24 (1.25), and for the high dose -4.11 (1.87). The ANOVA showed that the drug effect was significant ($F=28.23$; $df=2,22$; $P<0.001$). The reaction time data showed a trend consistent with the drowsiness estimates: for the placebo condition, mean RT (\pm sd) was 222.4 ms (5.9), for the low dose 225.4 (5.8) and for the high dose 234.6 (6.8). However the effect was not significant (from the ANOVA, $F=1.22$; $df=2,22$; $P>0.05$).

Discussion

The present experiments show that the tilt illusion is unaffected by either dopaminergic agonist or antagonist drugs within the dose ranges chosen. These results are in contrast to earlier work (Harris et al. 1983, 1986) in which we found that dopamine agonists increase and antagonists decrease the tilt aftereffect. On the other hand, the tilt illusion is increased by lorazepam, a drug which potentiates GABA-mediated neurotransmission, in a dose-dependent fashion. The tilt aftereffect was, by contrast, unaffected by lorazepam.

Some potential artefacts

It is interesting that despite their similar effects in increasing subjective drowsiness, the neuroleptics and the benzodiazepine have different effects on visual processes. This suggests that the visual effects are not simply a concomitant of the effects on alertness. Additional evidence against simple drowsiness effects underlying the results was obtained with maprotiline. Compared to lorazepam, subjective drowsiness ratings after maprotiline were similar. However, only lorazepam caused a change in the tilt illusion. It is clear, therefore, that the presence of drowsiness does not predict drug-induced changes in either the tilt illusion or the tilt aftereffect.

It might be argued that our failure to find significant changes in the TI with, say, haloperidol and in the TAE with lorazepam arose from some failure in techniques, which were not sensitive enough to detect drug-induced changes in perception. However, the findings that the techniques detect perceptual changes with other drugs, and that the drugs induce changes on other tests are evidence against this view.

In interpreting the results, we assume that similar processes underlie the tilt and motion aftereffects, even though they may operate on different neural channels. In general, this assumption is justified by the data, which suggest that the two aftereffects are similarly affected by the various drugs we have studied. An exception to this rule are the effects of nomifensine which enhances the TAE, but not the MAE, though the trends in the MAE data are similar (Harris et al. 1986). Our procedure for measuring the MAE does not allow us to distinguish between changes in neural adaptation and changes in criterion for the end of apparent motion (see Harris et al. 1986 for further discussion of these points). If nomifensine affected both of these, it might lead to the obtained result. Certainly, this result cannot be used

to argue for different processes underlying the MAE and TAE.

Neural processing and the mechanism of the illusions

How might the dissociation between the perceptual effects arise? There is an important difference between the procedures for obtaining TAEs and TIs which might lead to drugs (or other variations) having differential effects on the two illusions. This is the prolonged inspection of the adapting grating alone which is necessary to obtain the TAE. In the TI, both gratings are present simultaneously, and long inspection periods are not required. If we accept that lateral inhibition underlies both the TAE and TI, and follow Wallace (1975) in the simple assumptions that the lateral inhibition produced by a neuron is proportional to its excitation and that excitation and inhibition add linearly, then any manipulation which reduced excitation during adaptation (leaving excitation by the briefly viewed test grating relatively unaffected) would reduce the TAE. However, such a manipulation would not affect the TI, since the neurons responding to the "inducing" and "test" gratings would be equally affected and the relative strengths of their excitation and mutual inhibition would be unchanged. On the other hand, a manipulation which affected cortical lateral inhibition alone would affect both the TAE and the TI in the same way. Finally, a manipulation which changed the strength of excitation in one direction and of inhibition in the other direction would affect the TI, but its effects on the TAE would depend on the relative amounts of change in excitation and inhibition.

The physiological evidence strongly suggests that lateral inhibitory interactions which underlie orientation selectivity in the visual cortex are mediated by a GABA neurone (Sillito 1979; Somogyi et al. 1983). Thus the simplest explanation for the enhancement of the tilt illusion by lorazepam is an increase in lateral inhibition between cortical orientation detectors. However, the finding that the TAE and MAE are scarcely affected by lorazepam suggests that, if lateral inhibition underlies them, the drug may have more complicated effects, since if its sole effect were on cortical lateral inhibition then the aftereffects as well as the illusion would be affected. One possibility is that the drug reduces excitation in visual channels, as well as enhancing inhibition between them. If the lateral inhibition exerted by a neuron is proportional to its excitation (as assumed for a related illusion by Wallace 1975) then the potentially greater lateral inhibition during the adapting period of the TAE and MAE could be nullified by the reduction in excitation, so that little net change in aftereffect would occur. The finding (Harris and Phillipson, in preparation) that lorazepam reduces contrast sensitivity and threshold elevation for similar grating displays is consistent with this view.

Turning to the drugs which change dopaminergic activity, in summary we find that dopamine receptor blockers reduce the aftereffects, and nomifensine enhances the TAE (Harris et al. 1986), but have no significant effects on the tilt illusion. It is clear that one of our earlier suggestions, that the blockers reduce the TAE and MAE by reducing lateral inhibition in the cortex, is not supported. If this were the case then one would expect that the tilt illusion would also be reduced, but this was not found. Indeed, though insignificant, the tendency was for the drugs to have the opposite effect on the tilt illusion to that on the tilt

aftereffect. To follow the line of reasoning put forward to account for the lorazepam effect, these results suggest that this second class of drug changes excitation in visual channels rather than lateral inhibition between them. One result of this alteration may be an associated change in adaptation. The functional nature of the postulated change of excitation produced by the drugs cannot be further specified from the present evidence. Among the possibilities are a lowering of sensitivity across the entire contrast range, or a change of supra-threshold gain with no effects on absolute threshold. These questions are now under investigation by measuring contrast thresholds before and after adaptation to high contrast gratings, following administration of drugs.

Though speculative at present, the idea that the contrast gain of selectively activated visual channels may be reduced by neuroleptics, and thus involve dopaminergic mechanisms, is an attractive one. For example, a commonly reported symptom of schizophrenia is a change in brightness contrast (see, e.g. Chapman 1966). In a questionnaire study (Phillipson and Harris 1985), we found that, in a sample of 45 patients who reported visual distortions, abnormalities of brightness contrast were the most common symptom. Since changes in luminance contrast are known to influence the perception of other visual properties such as velocity (Thompson 1982; Harris 1982) or colour (Troscianko 1977) and to influence the size of the TAE (Parker 1972) the hypothesis that disorders of gain control (subjectively, a change of contrast) may underlie some visual distortions of schizophrenia seems worth pursuing further.

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