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Psychotropic Drug-Induced Transformations of Visual Space

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Abstract. It was found that ergotropic arousal-inducing drugs, such as psilocybin, a Ditrin®-type 'glycolate' and D-amphetamine, significantly lower human spatial distortion thresholds, i.e. these drugs *interfere* with counter-adaptation to optical distortion, or the intention to see the world undistorted. The trophotropic arousal-inducing chlorpromazine® on the other hand *promotes* such counter-adaptation, i.e. the optimization of visual information. The interference with optimization is independent of the rate at which the distorting stimulus is presented.

Optimization is regarded here as a cortical (perceptual-behavioral) interpretive process while interference with and promotion of the optimization are subcortical influences.

Optimization of Visual Information under Drug-Induced Ergotropic (or Hyper-) and Trophotropic (or Hypo-) Arousal¹

The continuous wearing of prism spectacles induces a variety of visual distortions which, however, gradually disappear (3). Such 'adaptation' to distortion may be impeded by classical hallucinogens such as psilocybin², Ditrin®-type anticholinergic psychotomimetics — here called 'glycolates' — and the sympathomimetic D-amphetamine. 'Adaptation', on the other hand, may be enhanced through phenothiazine-type tranquilizers such as chlorpromazine®.

¹ Ergotropic arousal denotes according to Hess (1), behavior patterns that are preparatory for positive action and characterized by increased sympathetic activity and an activated psychic state. The trophotropic effect, on the other hand, consists of increased parasympathetic discharges, relaxation of the striated muscles, cortical synchronization and sleep (2).

² The cross-tolerance between psilocybin, LSD and mescaline (4, 5), as well as their characteristic square-wave pattern of saccadic eye-movement (6), mark these drugs as *the* psychotomimetic, psychodysleptic or hallucinogenic drugs. It is implied, therefore, that any state which can be elicited by one of these drugs, can be duplicated by the others as well.

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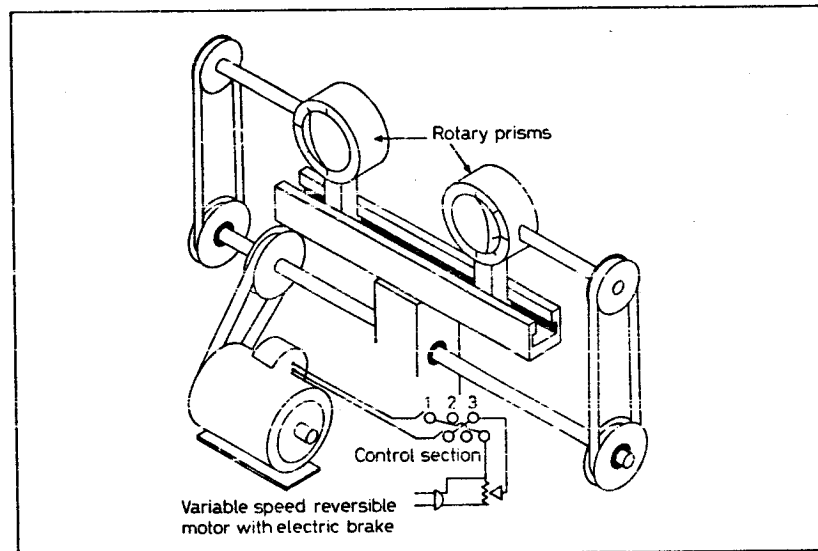


Fig. 1. Scheme of apparatus for the rate-controlled induction and measurement of SDT.

This paper explores and defines certain variables which appear to control drug-induced inhibition or enhancement of the adaptation phenomenon.

An instrument was developed to circumvent the risks inherent in the wearing of distorting prism spectacles while under drug-influence and to enable us to quantify rapid changes of the adaptation phenomenon (fig. 1). The main feature of this instrument is a pair of synchronously (motor-) driven counter-rotating prisms. Through these prisms, which can be driven at selected rates, the subject views a horizontal 15×7 cm black line at a 40-cm distance against a high-contrast white background of 85 cd/m^2 .

Distortions Induced at Constant Rate

These experiments were performed by producing the distorting optical stimulus at a constant rate of 5 prism diopters (Δ) per second. The just-noticeable bending of the black line marked a subject's 'spatial-distortion threshold' (SDT) expressed in Δ . Note that, for small angles, $1\Delta = 0.6^\circ$.

Fifteen Ohio State University student volunteers with a median age of 23 years and a functional visual acuity of at least 20/20 at the task-distance were studied prior to (T_1), 60 min after (T_2), 110 min after, i.e. at drug-peak (T_3), and 280 min after (T_4) the oral ingestion of $160 \mu\text{g/kg}$ psilocybin. A mean Δ -value was computed at T_1 , T_2 , T_3 , and T_4 from six determinations.

The results show that psilocybin maximally inhibits readaptation at drug-peak (T_3) by lowering the SDT (7). Similar results have been found in an independent study using a Ditrane®-type anticholinergic psychotomimetic drug (a 'glycolate') as well as our experimental design and instrumentation (8).

The extent to which the SDT is lowered, however, is largely a function of the subject's 'stability' or 'variability'. Stability is observed here by the consistently small standard deviations (SD) which a subject displays on a variety of perceptual tasks and behavioral tests without any drug, whereas 'variable' subjects display large and variable SDs on a yet-unlimited variety of perceptual tasks and behavioral tests without drugs (9, 10).

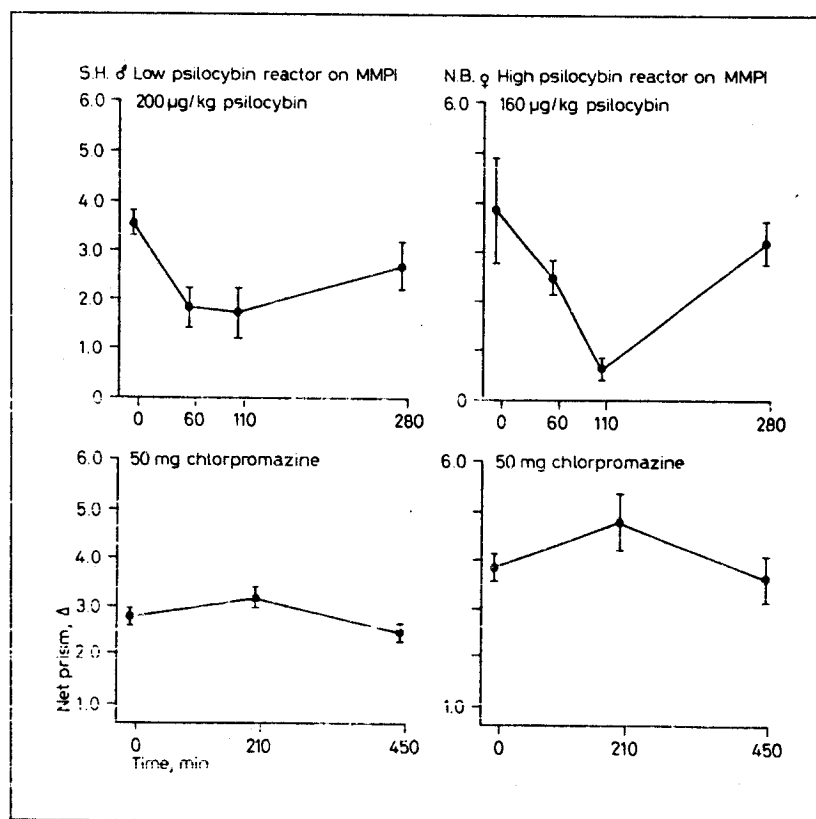
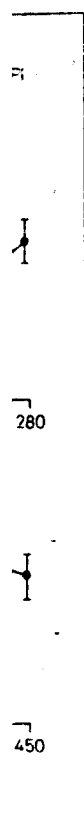


Fig. 2. SDTs of a 'stable' (S.H.) and a 'variable' (N.B.) subject after the oral administration of 160 μg/kg of the hallucinogenic drug psilocybin (above) and the tranquilizer chlorpromazine (below). Each point represents the mean of 6 measurements; the SDs are indicated by the vertical bars. Stability or variability of a subject is defined here by the SD at 0 time (T_1).

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Note that in a 'variable' subject such as N.B.♀ at T_3 , i.e. at drug-peak, 110 min after the administration of 150 $\mu\text{g/kg}$ psilocybin, the ergotropic arousal-induced interference with adaptation is maximal. Very little prism power — only 0.7 Δ , i.e. 15–20 % of the pre-drug value — is needed to just-noticeably bend the horizontal line-target (fig. 2). In the stable subject S.H.♂, however, the ergotropic arousal-induced maximal interference with adaptation at T_3 is relatively small: 1.7 Δ are still required, i.e. at least 50 % of the pre-drug value, for the just-noticeable distortion of the line-target (fig. 2).

Another important point is that the small SD of a 'stable' subject at 0-time or T_1 (see upper left in fig. 2) is an indicator of his greater stability against the drug-induced interference with adaptation at drug-peak, i.e. 110 min after psilocybin ingestion (T_3). On the other hand, the large SD of a 'variable' subject at 0-time or T_1 (see upper right of fig. 2) indicates in advance that he will become more susceptible at drug-peak (i.e. 110 min after psilocybin-ingestion or T_3) to the drug-effect, resulting in an extensive lowering of his SDT.

If psychotomimetic drugs such as psilocybin or a 'glycolate' significantly lower SDTs — especially in 'variable' subjects — what is then the effect of a tranquilizer?

We have chosen the same two representative 'stable' and 'variable' subjects (on the left and right half of fig. 2) to illustrate the (phenothiazine tranquilizer) chlorpromazine-induced trend to raise SDTs. It can be stated that the two antagonistic types of drugs, the psychotomimetics and the tranquilizers, also 'behave' antagonistically, i.e. they lower and raise SDTs, respectively. The chlorpromazine-induced increase of the SDT appears to be more exaggerated in a 'variable' subject such as N.B.♀ (on the right of fig. 2), in whom the SDT-lowering effect of psilocybin was also more pronounced.

An analogous subject-dependent influence of psilocybin on behavior was found when drug-induced behavioral changes in fifteen subjects were measured with the Minnesota Multiphasic Personality Inventory (MMPI). Specifically, behavioral 'stability' or 'variability' — measured with a score obtained from the sum of the squared ($T_3 - T_1$) differences (D^2) of 13 K-corrected T scores, across sexes of the MMPI — paralleled perceptual 'stability' or 'variability' (4) — the Spearman rank-order correlation coefficient $r_s = 0.64$ ($p < 0.01$) (9).

Psilocybin can then considerably lower SDTs and alter MMPI-measured behavior in 'variable' subjects, whereas 'stable' subjects are less affected by the drug.

The action of psilocybin and that of a 'glycolate' on SDTs is not restricted to these two types of psychotomimetics. Fifteen mg of the sympathomimetic *D*-amphetamine can also lower the SDT (fig. 3).

It appears, therefore, that excitatory drugs in general lower SDTs, and particularly so in 'variable' subjects. There is, however, the uncommon combination of a 'stable' subject with an exceptionally low SDT prior to drug-ingestion. The

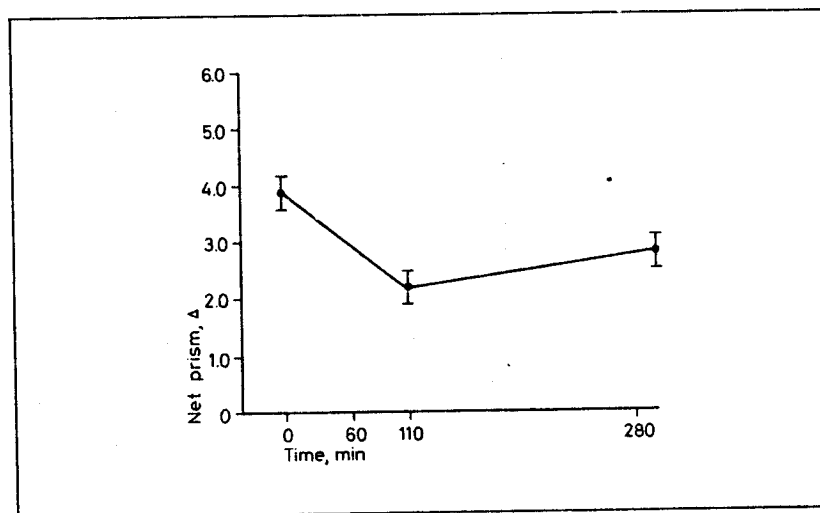


Fig. 3. SDTs of a 'stable' subject (B.K.δ) after the oral administration of 15 mg *D*-amphetamine. Each point represents the mean of 6 measurements; the SDs are indicated by the vertical bars.

already low SDT in such a person apparently cannot be further lowered by 160 $\mu\text{g/kg}$ psilocybin as observed in one among 17 cases. Nevertheless, the trend of change with respect to the SD at T_1 , T_2 , T_3 and T_4 is almost identical to the pattern displayed by our stable subject on the upper left of figure 2.

Distortion Induced at Several Selected Rates

Whether optimal 'adaptation' in response to optically induced distortions depends on the rate at which the distortion is presented, has not yet been investigated. To explore this possibility we have converted our prism drive system in such a way that the distorting stimulus could be presented throughout a 0.5–10.0 prism diopter-per-second (Δsec^{-1}) range. Each individual SDT as well as the rate at which the SDT was induced, was then automatically graphed for subsequent analysis by a potentiometric strip recorder (fig. 4).

Using a sample of 16 college-age volunteers and an oral dose of 160 $\mu\text{g/kg}$ psilocybin, the same black-line target was presented to each subject as described in the first part of this paper, but at 12 different rates within 6 min at T_1 , T_2 , T_3 and T_4 . The 12 rates were uniformly distributed throughout the 0.5–10 Δsec^{-1} range and were offered in a random fashion.

The SDTs in Δ from each potentiometric record were then re-plotted as a function of rate of distorting stimulus presentation in Δsec^{-1} . Both the slope

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and intercept values derived from a regression analysis of each of these functions were then tested for covariance under pre-drug, drug-peak and post-drug conditions using Pearson product moment correlation analysis.

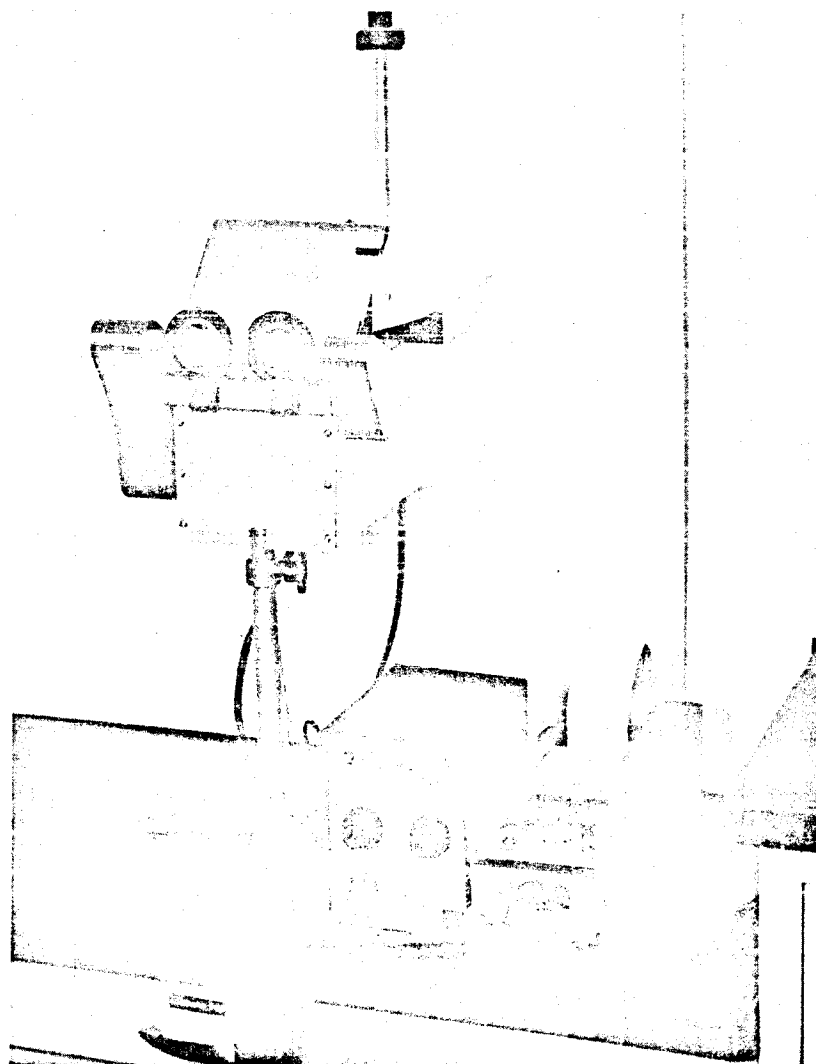


Fig. 4. Apparatus for the variable rate-controlled induction and measurement of SDTs. The seated subject views through motor-driven counter-rotating prisms a horizontal black line, reflected in a 45° front-surface mirror. The just-noticeable bending of the line indicates the subject's SDT in prism diopters. The variable prism speed control and the linear potentiometric recorder are shown to the right.

Although, with an increase in the rate of presentation, there is a proportionate raise in the SDT for each subject, this raise is not measurably affected by psilocybin under our experimental conditions. *We conclude, therefore, that the hallucinogenic drug psilocybin exerts an ergotropic arousal-induced lowering of the SDT – or, in other words, the drug interferes with 'adaptation' to optical distortions – but this interference is apparently independent of the rate at which the distortion is presented.*

Discussion

The most significant finding of our study is the ergotropic arousal-induced lowering of the SDT. Drugs which elicit ergotropic arousal, i.e. a sympathetic (excitatory) state, interfere with 'adaptation' to optically provoked distortions. This interference is most marked in 'variable' subjects, i.e. those who display variable SDs on a variety of perceptual tests and/or behavioral tasks. 'Stability' or 'variability' (i.e. the SD itself) follows a log-normal distribution in a population, as we have found, and therefore, 'stable' subjects are rather uncommon since 'variable' or unpredictable subjects constitute the middle range of the distribution representing about two-thirds of the general population. It is these 'variable' subjects who are more affected by ergotropic arousal-inducing drugs than the 'stable' subjects represented by the tail of the log-normal distribution curve.

Trophotropic arousal-inducing drugs, on the other hand, seem to increase SDTs and thus enhance the phenomenon of 'adaptation' insofar as we are able to extrapolate from our pilot studies involving chlorpromazine. The 'adaptation'-enhancing effect of this tranquilizer is again most pronounced in 'variable' subjects.

What about the possible practical implications of these results? The most common optical distortions result from the peripheral curvature of windshields in automobiles and aircraft. Windshield curvature, indeed, can induce topographical distortions of the environment. Similarly, any corrective lenses with high ametropic or prismatic components, sighting devices incorporating prism systems such as periscopes, optical rangefinders, etc., may also produce distortions.

In the light of our data, therefore, the excitatory hallucinogenic drugs which interfere with 'adaptation' to optically induced distortions are contraindicated whenever performance depends on such distortion-inducing systems. Tranquilizers, on the other hand, although they do not interfere with 'adaptation', are not recommended either, but for other reasons. Tranquilizers induce an underestimation of chronological time (11) (e.g., a tranquilized subject arrives late to an appointment, if without a watch) and may thus impair or delay decision-making.

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Our finding, that the interference of the excitatory hallucinogenic drugs with 'adaptation' to optically induced distortions is independent of the rate at which the distortion is presented, has further implications. The interference effect — especially in 'variable' subjects, i.e. in two-thirds of the population — remains significant at any rate of presentation and, thus, it can be safely stated that hallucinogenic drugs are 'unsafe at any speed'!

But what is that which is being distorted, and furthermore, what is the meaning of 'adaptation'? We suggest that what is being distorted is a constancy of relations (12) representing specific perceptual information. *Informational constancies* are in fact the framework of our spatio-temporal world. 'Adaptation', then is an attempt to counter-adapt with the aim of restoring the status quo through optimization of available information.

What can be said about the nature of this self-deceptive restoration of constancies? Apparently, in spite of the distorted image on the retina, the subject 'sees' a re-stored image, i.e. an image congruent with the subject's past experience and expectations. This re-storative procedure, a cortical optimization of perceptual information in the interest of survival, could be conceptualized, at least in the case of vision, as a coordinate transformation process.

It is however, arbitrary to detach the *conceptual* from the *perceptual* process. Conceptual factors are at least as important in maintaining the stability of the world, as exemplified by *Wittreich's* data which show that an aniseikonic lens-induced distortion of the human figure is differentially resisted according to the degree of familiarity of the observer with the figure (13, 14). Specifically, less distortion, i.e. more readaptation, was always reported in a spouse than in a stranger; less in a mutilated than in a normal figure; and navy recruits reported less distortion of authority figures than of non-authorities. Children describe less distortion in a parent than in a stranger and, the most important of all, the younger the child, the less developed is his ability to readapt to distortions, emphasizing the gradually learned perceptual-conceptual nature of constancies. Even phantom sensations, which are readaptation phenomena maintaining the constancy of the self (15), do not appear if amputation is performed before the age of four years (16).

The re-storing of distorted reality is in fact an imaginary re-interpretation of 'reality as it should be'. Based on past experience and expectations, the image of distorted reality is re-stored through a 'normal' visual hallucinatory experience, a vision in imagination. Evidently, the optimization proceeds in the mental dimension, irrespective of and directed against the distorting stimuli of the physical dimension. But hallucinations — like dreams — must not be necessarily pathological. According to our definition, both dreams and hallucinations are experiences of intense inner sensations with concomitantly inhibited or even blocked intention and ability to verify those sensations through voluntary motor performance (17, 18). Indeed, there is no need for the verification of the re-storing experi-

ence, since only perceptual experiences (not dreams and hallucinations) can be verified through voluntary motor performance. The only verification of the 'adaptation' phenomenon is through past experience and expectations.

It is tempting to juxtapose at this point the *cortical* optimization process with the phenomenon of geometrical illusions. There is no optimization involved in geometrical illusions since the mechanisms involved in these illusions are anchored in *subcortical* structures, most probably in the retina itself (19). After having been exposed to (or brainwashed by) the subjectivizing influences of epistemological dualism, Locke's primary and secondary qualities, Müller's specific nerve energies and the causal theory of perception, we may say that a geometrical illusion is probably in the *subcortical* (retinal) structure of the beholder, whereas the re-stored, optimized image of an optical distortion is a function of the *cortical* interpretation of the beholder.

If experience is the perceptual-behavioral or cortical interpretation of subcortical activity, what is the nature of the relation between this symbolic interpretation and subcortical activity – the biological substratum – it interprets? We submit that, at the arousal levels of daily routine, and even during moderately aroused states, the perceptual-behavioral interpretation is to a great extent independent of, or dissociated from, the subcortical activity it interprets (10, 15, 20, 21, 22). But when we depart from this world of daily routine along the perception-hallucination continuum of increasing levels of ergotropic arousal, or along the perception-meditation continuum of increasing trophotropic arousal, perception-behavior becomes more and more dependent on the biological substratum (which generates it). Such loss of freedom results in an increased stereotypy which the individual may interpret as inspiration, 'déjà vu', 'flash-back' or possession. Increased stereotypy means that – due to the enmeshment of cortical and subcortical activities – the individual's repertoire of available symbolic interpretations gradually diminishes with increasing arousal (18, 23).

In view of the above, a concomitant study of geometric illusions and SDTs under the influence of particular drugs can assist in the elucidation of the ergotropic and trophotropic arousal induced interdependence of cortical and subcortical structures.

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