

DRUGS AND PERSONALITY

III. THE EFFECTS OF STIMULANT AND DEPRESSANT DRUGS ON VISUAL AFTER-EFFECTS

By

H. J. EYSENCK

H. HOLLAND

and

D. S. TROUTON*

Institute of Psychiatry (Maudsley Hospital), University of London

It is a well-known principle in psychology that the effects of sensory stimulation do not cease when the stimulus itself ceases. The so-called visual after-images are probably the best known of these after-effects, but almost equally well known are those induced by a rotating spiral (11). If a normal person fixates the centre of the rotating spiral for a period of time, and if then the motion of this spiral is suddenly stopped, an after-sensation of movement is well nigh universal. This after-sensation is opposite to the original motion in direction, just as the after-image produced by the fixation of a colour is usually the complementary colour to that which caused the original perception. There is little doubt that while peripheral factors play some part in these after-effects, central features also play a part (9), and it becomes tempting, therefore, to use phenomena of this type in attempts to verify or disprove the drug action hypotheses advanced by one of us in a previous paper (2). This hypothesis stated that stimulant drugs increased excitatory potential and decreased inhibitory potential, while depressant drugs decreased excitatory potential and increased inhibitory potential. The question now arises as to how this general postulate can be applied to the phenomena under discussion.

We are handicapped from the outset by the fact that the explanation of these after-effects is not in fact known. There are many different hypotheses, but it cannot be said that any of these are at all widely accepted (11). In the absence of a detailed theory, we may perhaps argue as follows. Underlying the perceptual after-effect, we must postulate some kind of neurological substratum, i.e. the passage of neural currents across the synapses. Nothing can usefully be said about the precise nature of this hypothetical sub-stratum, but few people would probably doubt the dependence of psychological phenomena on neurophysiological events. Granted such a basis, it follows immediately from our knowledge of the processes of satiation and inhibition that these neural currents will produce inhibitory potentials which will tend to decrease the strength of these currents and finally stop them altogether (7, 8).

If this very rough and ready model is at all along the right lines then we would expect extraverts, hysterics and brain-damaged people to have very short after-effects, while introverts and dysthymics would have particularly long after-effects. This follows from the postulate advanced in the previous article (1) linking extraversion with excessive inhibition and introversion with

* We are indebted to the Bethlem Royal and Maudsley Hospital Research Fund for support which made this study possible.

excessive excitation. It would also follow that depressant drugs should decrease the length of the after-effect, while stimulant drugs should increase the length of the after-effect (4). We therefore have a variety of ways in which various aspects of our general hypothesis can be tested. The only relevant paper we have been able to find in the literature is an article by Klein and Krech (6) in which they refer to an unpublished paper by Klein. Apparently Klein found that "where persistence of after-image was measured as a function of the duration of stimulus-exposure, it appeared that for longer exposures the duration of the after-images of brain-injured fell off significantly as compared with non-brain-injured. This could be interpreted to mean that among brain-injured neural activity in regions which had previously been exposed to prolonged excitation is 'dampened' as compared to 'normals', i.e. consequent upon the same amount of original excitation there is a greater degree of satiation in the brain-injured than in the non-brain-injured. That satiation in the brain-injured is not only greater in extent than in 'normals', but that it also persists for a longer time is suggested by another finding of Klein's: the rate of decrease in after-image duration upon repeated exposures was more rapid in the brain-damaged than in the controls. In general, then, Klein's studies are congruent with the hypothesis that in the brain-injured successive, prolonged exposure to stimulation induces *satiation* attributes . . .". It appears then that with respect to after-images, brain-damaged people, in line with our prediction, produce shorter after-effects than normals.

Price and Deabler (10) and Gallese (5) supply evidence showing that the brain-damaged are also inferior to normals with respect to the after-effects induced by the rotating spiral. Price and Deabler tested 40 normals, 40 non-organic psychotics, and 120 brain-damaged subjects on the spiral after-effect, giving 4 separate tests and simply determining whether or not an after-effect was perceived. (This method, which was also followed by Gallese, is very rough and ready, and should almost certainly be replaced in future studies by a more systematic exploration of the length of after-effect as a function of length of stimulation. However, the fact that positive results can be achieved even with this very crude technique suggests that the differences are very pronounced indeed.) Their results are given in Table I below, in which are reported the percentages in the various groups which saw after-effects on 0, 1, 2, 3, or 4 occasions; it will be obvious that the brain-damaged group is very significantly differentiated from the normal and functional groups.

TABLE I

			0	1	2	3	4
			Per cent.	Per cent.	Per cent.	Per cent.	Per cent.
Normals	0	2.5	0	5	92.5
Functionals	0	0	2.5	2.5	95
Brain-damaged	60	10	20	8	2

These very striking differences were to some extent verified in the study by Gallese, who scored his test in terms of a cut between 2 or less, and 3 or more reports of seen after-images, calling the former an "organic" and the latter a "normal" score. In 30 normal subjects, he found exclusively normal reactions, while in 41 schizophrenics he found 95 per cent. of normal reactions, thus substantiating previous findings that this test does not differentiate between normals and functional patients. An organic group consisting of 47 patients suffering from disorders other than those diagnosed alcoholic and convulsive

disorders showed only 34 per cent. of normal reactions; another organic group consisting of 50 patients suffering from alcoholic and convulsive disorders showed 72 per cent. of normal reactions. Twelve lobotomized schizophrenics showed only normal reactions. The retest reliability of the score was found to be high, the fourfold point correlation between first and second testing on 34 organic patients being .84, for two different examiners.

Gallese concludes that "with this method of inquiry, and of scoring, the test almost always indicates organicity when organic scores are obtained, although the converse is not true". He adds the observation that "it is . . . the author's belief that among the organics who obtained high scores the duration of the negative after-effect was considerably less than among the non-organics". Altogether, there appears to be considerable support for our deduction.

Little evidence is available with respect to the relationship of the length of after-effects and extraversion-introversion. In one small study 17 university students were administered a personality questionnaire and also tested on the Archimedes Spiral using 4 different periods of stimulation (10 seconds, 30 seconds, 50 seconds and two periods of 100 seconds, one of them preceding, the other following, the other three tests). Length of after-effect was measured in terms of the reports of the subjects as to when they ceased to see the after-effects. Figure 1 shows the mean scores of the 4 most extraverted and the 4 most introverted subjects; it will be seen that as predicted the introverts show a longer after-effect than do extraverts.

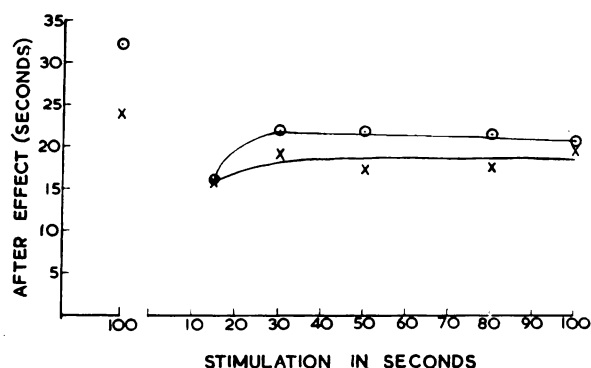


FIG. 1.—After-effect in seconds of stimulation by rotating spiral for groups of extraverts (crosses) and introverts (circles). Stimulation for 100 seconds was followed by a lengthy pause and then by successive stimulation for varying periods as indicated on the abscissa.

The spiral used in this experiment was a 4-throw spiral of 180°, this type having proved the most successful in several preliminary experiments. The spiral is 8½ inches in diameter, subtending a visual angle of 6½ degrees. It is constructed of thick rigid cardboard and is rotated by an electric motor at a speed of 100 r.p.m. This speed was set initially by Strobe lamp and checked periodically, any slight variation being controlled by a variable resistance built into the circuit. (A photograph of the apparatus and spiral is given elsewhere (4).)

The disk can be rotated in a clockwise or an anti-clockwise direction depending on the position of a reversing switch. Illumination for the disk was provided by an Osram 100-watt pearl lamp, mounted 40 inches from the centre of the disk and 12 inches above it, thus being in the same direction but above the subject's line of regard. A fixation point was provided by the central, brass, locking nut of the spiral, which was polished by buffing.

We must now turn to the drug experiment. Each subject was given three treatments, on three different days; order of treatments was counter-balanced in the form of a double Latin Square.

1. D-amphetamine Sulphate, 10 mg.
2. Sodium Amylobarbitone, grains $4\frac{1}{2}$.
3. Placebo.

Both drugs and placebo were in identically appearing capsule form and taken orally. Owing to the differential absorption rates of the drugs, testing of treatments 2 and 3 began one hour after administration and testing of treatment 1 began $1\frac{1}{2}$ hours after administration. Subjects were instructed to rest until required.

TESTING PROCEDURE

The subject was seated on a hardwood chair, the front legs of which were six feet away from the spiral. As already noted, the spiral was illuminated and this was the only illumination in an otherwise completely darkened room. The following instructions were then read: "In a few moments I am going to rotate this disc and shall do so for one minute. During the time it is rotating I want you to keep your eyes fixed on that brass nut you see shining in the middle. At the end of one minute I shall stop the disc and you will see an after-effect having the appearance of a contraction or expansion coupled with a rotation in the opposite direction from that in which the disc was previously turning. I want you to merely say the word 'now' as soon as this expansion or contraction completely ceases."

When it was quite certain that the subject understood the instructions the motor rotating the disc was switched on and continued for a period of one minute (timing being done by stop-watch). At this point the motor was switched off and the stop-watch allowed to continue until the arranged signal was given. There followed a one-minute rest interval, during which the reversing switch was turned from "forward" to "reverse" and the second trial then began. The whole experiment consisted of four such trials.

Scoring is of the simplest kind; the time on each trial is taken in seconds and the mean of the four trials constitutes the score of that subject under that treatment.

SUBJECTS

The total N in this experiment was six, being, with one exception, all postgraduate students of psychology. The exception was a senior technician in the Department of Psychology. The ages of the sample ranged between 25 and 39, with a mean of 29.5. There were five men and one woman.

RESULTS

The prediction in this case was that treatment 2 (sodium amylobarbitone), being a depressant and thus an extraverting drug, would shorten the duration of the persistence of the after-effect relative to the "natural" level as assessed under treatment 3 (placebo), and that treatment 1 (d-amphetamine sulphate), a central excitant and thus an introverting drug, would lengthen it. Table II

TABLE II

Source	Degree of Freedom	S. Squares	Mean Squares	F	P
Replication	3	125.11	41.70	2.756	$= < .05$
People	5	2003.33	400.67	26.482	$= < .001$
Drugs	2	376.58	188.29	12.445	$= < .001$
Days	2	45.75	22.87	1.512	N.S.
Replication/Drugs ..	6	28.64	4.77	—	N.S.
Replication/People ..	15	213.56	14.24	—	N.S.
Residual	38	575.03	15.13	—	
Total	71	3368.00			

shows the results of an analysis of variance carried out to assess the significance of the differences between the scores. It will be seen that three significant F ratios emerge, namely, those for "replications", "people" and "drugs", the other main effect and the interactions remaining insignificant. Figure 2 shows the results in diagrammatic form.

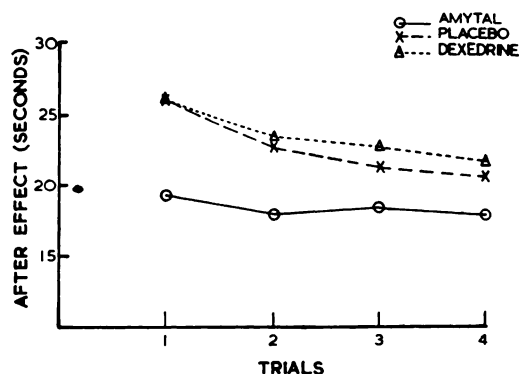


FIG. 2.—After-effects in seconds of stimulation by rotating spiral for group of subjects under drug treatment.

The fact that the replication term of the analysis reaches significance at the 5 per cent. level of confidence, coupled with the non-significant interaction of "replication" with "people" suggests that a consistent downward trend from trial to trial in the duration of the after-effect (23.83, 21.38, 21.16, and 20.27), might be interpreted as some form of practice effect, or that insufficient time was allowed between trials to permit the complete dissipation of inhibitory potential. The two remaining significant differences support the hypotheses tested, thus a P of .001 confirms the main theoretical thesis that considerable differences exist between people (i.e. subjects), and finally a P of .001 between drugs confirms the main drug hypothesis.

Further information was obtained by running t tests between treatments. Significant differences were found to exist between treatments 1 and 2 (i.e. dexedrine and amytal) and between 2 and 3 (i.e. amytal and placebo), both these differences invalidating the null hypothesis with a $P = .001$. The differences between treatments 1 and 3 (dexedrine and placebo) failed to achieve a level of significance which could be regarded as adequate.

This is an interesting result in that it supported a subjective opinion of the writers that dexedrine did not appear to have the same effect as an excitant, as

amytal had as a depressant, with respect to the general behaviour of the subjects. There may be at least two reasons for this. The experimental design, though allowing for a differential absorption rate, may not have permitted sufficient time for the amphetamine to have its maximal effect. In a previous paper (3), it has been shown that "time since administration" has an important bearing on the effectiveness of this drug.

The overall results of the experiment are clearly favourable to our main hypothesis. If the experiment were to be repeated it might be advisable to test the effects of a stimulant drug after allowing a rather long period of time to elapse between administration and test.

SUMMARY

The prediction was made that stimulant drugs would increase the duration of visual after-effects while depressant drugs would have the opposite effect. Using the length of after-effects produced by a rotating spiral as our measure, six subjects were tested under three conditions, each on separate occasions. The three conditions were: (1) After administration of dextedrine; (2) After administration of sodium amytal; (3) After administration of a placebo.

The results showed that the sodium amytal had a highly significant depressant effect on the phenomenon studied, as compared with the placebo and dextedrine conditions. Dextedrine, while acting in the predicted direction, produced an effect which was not strong enough to reach full statistical significance.

REFERENCES

1. EYSENCK, H. J., "A dynamic theory of anxiety and hysteria", *J. Ment. Sci.*, 1955, **101**, 28-51.
2. *Idem*, "Drugs and personality: I. Theory and methodology", *J. Ment. Sci.*, 1957, **103**, 119-131.
3. *Idem*, CASEY, S., and TROUTON, D., "Drugs and personality: II. The effects of stimulant and depressant drugs on continuous work", *J. Ment. Sci.*, 1957, **103**, 645.
4. EYSENCK, H. J., *The dynamics of anxiety and hysteria*, 1957. London: Routledge & Kegan Paul.
5. GALLESE, A. J., "Spiral after-effect as a test of organic brain damage", *J. clin. Psychol.*, 1956, **12**, 254-258.
6. KLEIN, G. S., and KRECH, D., "Cortical conductivity in the brain injured", *J. Personality*, 1952, **21**, 118-148.
7. KOHLER, W., and WALLACH, H., "Figural after-effects", *Proc. Amer. Phil. Soc.*, 1944, **88**, 265-357.
8. OSGOOD, C. E., *Method and theory in experimental psychology*, 1953. New York: Oxford University Press.
9. POPOV, CATHERINE, *Contribution à l'étude du mécanisme d'élaboration des connexions corticales*, 1955. Paris: Parnasse.
10. PRICE, A. C., and DEABLER, H. L., "Diagnosis of organicity by means of the spiral after-effect", *J. consult. Psychol.*, 1955, **19**, 299-302.
11. WOHLGLMUTH, A., "On the after-effect of seen movement", *Brit. J. Psychol., Monogr. Suppl.*, 1911, **1**.