DRUGS AND PERSONALITY

V. THE EFFECTS OF STIMULANT AND DEPRESSANT DRUGS ON THE SUPPRESSION OF THE PRIMARY VISUAL STIMULUS

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IN a previous paper in this series (3), a brief discussion has been given of the theoretical reasons for anticipating that depressant drugs would shorten the perceptual after-effects of visual stimuli, and stimulant drugs would lengthen these effects. The present paper is concerned with a related type of prediction, also concerned with certain visual after-effects. The phenomenon in question was first observed by Bidwell (1, 2); it has not received much study, unfortunately, in spite of its great theoretical interest. The phenomenon is simply this. If a beam of red light (the primary stimulus, or S_p) is thrown into the eye of a person having normal colour vision, that person will report a red sensation, followed after the extinction of the light source by a series of after-images, usually in the complementary colour. The theory governing the appearance of the after-image is conventionally a photochemical one; the S_p produces certain effects in the retina which are opposite in direction for complementary colours and which are reversed when the S_p ceases to act on the retina. This reversal is perceived as the complementary after-image. Bidwell discovered that when the S_p is shown only for a relatively short period of time, say 20 milliseconds, and is immediately followed by a neutral stimulus, such as a beam of white light, then the S is not perceived at all, but only the complementary after-image. In other words, a red stimulus is perceived as green, a green stimulus as red. It is this perception of a non-existent stimulus which caused the phenomenon to be of such interest to psychologists.

No theory appears to have been suggested in the literature to account for this phenomenon, and before making any kind of deduction from our general drug and personality theory (4) on this suppression of the primary visual stimulus, we must suggest in outline at least a possible explanation of the phenomenon. This explanation makes use of the concept of pre-excitatory inhibition (6), i.e. the well-known fact that the excitatory phase of a visual stimulus is preceded by an exceedingly rapid inhibitory phase. This inhibitory phase "wipes off" the after-effects of preceding stimulation, as it were, and makes possible a greater distinctness of successive visual stimuli. Theories of this kind have in the past been applied to the phenomena of visual flicker, and it is worthy of note that correlations have been found between CFF and measures of primary visual stimulus suppression thresholds (7).

Our theory, then, accounts for the observed phenomena as follows. The red stimulus light sets up two effects. In the first place, it sets in motion a photochemical reaction which goes on as long as the stimulus continues; when

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the stimulus ceases, this photochemical reaction is reversed. In the second place, the S_p sets up pre-excitatory inhibition (which is irrelevant to our theory), followed by a somewhat more slowly growing excitatory potential. This excitatory potential would be perceived in due course as *red*, but before that can happen the S_p is cut off, and followed by the white light. The cessation of the red stimulus produces post-excitatory inhibition, and the onset of the white light produces pre-excitatory inhibition; these inhibition effects make it impossible for the red to rise above the threshold of a sensation. Thus the failure of the primary visual stimulus to be perceived is explained.

The occurrence of the complementary visual after-effect is even easier to account for. When the red stimulus ceases to stimulate the retina, the photochemical process is reversed; this reversal, in accordance with the classical theory, is perceived as *green*, the complementary colour. As this reversal takes place *after* the post-excitatory inhibition due to the red light, and the pre-excitatory inhibition due to the white light have run their course, the green after-image is perceived without difficulty, although the simultaneous excitation produced by the white light somewhat attenuates its brilliance.

Given that this somewhat speculative hypothesis* is along the right lines, we may now deduce from our general drug hypothesis that depressant drugs will increase the inhibitory effects involved in the suppression of the primary visual stimulus, while stimulant drugs will decrease the inhibitory effects involved in the suppression of the primary visual stimulus. Thus the drugs in question should influence the time- and brightness-thresholds involved in the production of this phenomenon in opposite and predictable ways. The experiment here reported was carried out in an attempt to subject this hypothesis to an experimental check.

The Experiment

Six subjects were used in the experiment, each being exposed to three drug treatments on three different days. Treatments were administered according to a pre-arranged scheme, making use of a double Latin Square design; in this way sequence effects were controlled and eliminated from the main comparisons. A description of the subjects has been given in an earlier paper, and the selection and dosage of the drugs used has been discussed there also (3). Depressant, stimulant, and placebo were all given in capsule form, identical capsules being used.

The apparatus used for the production of the visual stimuli has been described in detail, and shown photographically, elsewhere (5). It consists of a light source which throws a shaft of white light through a red filter (Ilford No. 609 Spectrum Deep Red Filter) into a viewing tube through which the subject inspects the stimulus monocularly. The beam of light is interrupted by a rotating disc, into which a small sector has been cut; this sector allows the red beam to pass for a variable period, depending on the speed of rotation and the size of the sector, before it is interrupted again by the solid disc. Pasted on to the disc, on the subject's side, is a piece of white paper illuminated by a white

* It is important to be clear about the precise parts of this theory which are speculative. There can be little doubt about the inhibitory effects of the white stimulus following the red; these inhibitory effects are apparent *only* and *always* when a white stimulus of appropriate brightness and duration is administered after the red. What is speculative is the additional hypothesis that Granit's demonstration of the existence of pre-excitatory inhibition effects in the analysis of ERG records may be relevant to the observed relationships. This additional hypothesis is not necessary to our argument, and need not be accepted. Direct experimental evidence regarding the effects of drugs on the ERG is urgently needed to confirm or infirm this extension of our theory.

light source, giving a luminance of approximately 50 mL. The reflected light from this paper, which immediately follows the red light, constitutes the source of the hypothetical inhibition phenomena which eliminate the perception of the red light; omission to provide for the presence of the white light after the red eliminates the phenomenon under discussion completely.

Two experiments were carried out. In both experiments the subjects had to vary the brightness of the red light, by means of a variac, in such a way that the short 20 msec. red flash was reduced in intensity until no more red at all was perceived in the viewing tube and the coloured flash was seen as being entirely green. The arbitrary variac setting number corresponding to the perception of "optimum green" was taken as the score.

In the first experiment, the 20 msec. red flash was followed by a 125 msec. white flash. For experimental purposes unconnected with the present study, a 225 msec. white flash was introduced *before* the red flash, separated from it by a variable length of no stimulation (black). These white flashes preceding the red flash at different intervals had no effect on the phenomenon, and may therefore be disregarded. The results of the experiment are shown in Figure 1. On the

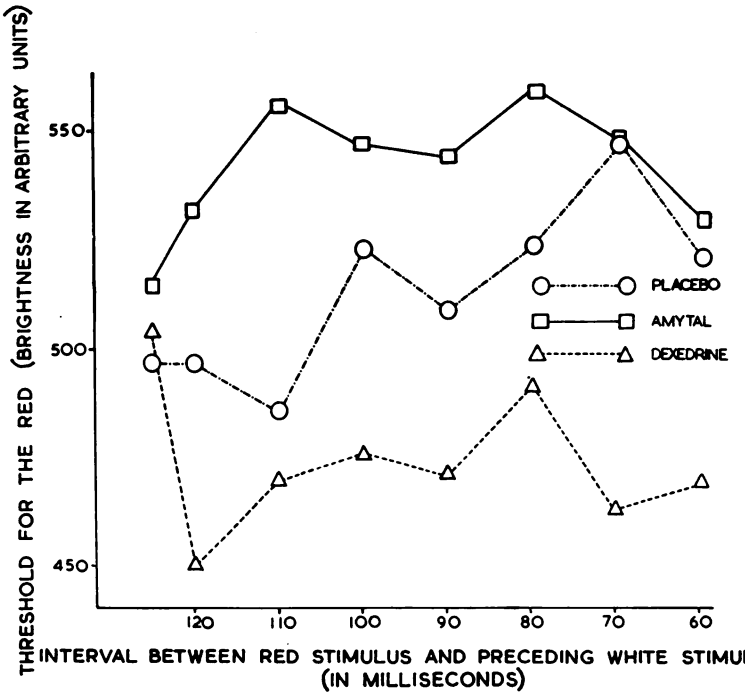

FIG. 1.—Threshold values for three drug groups plotted against interval between red stimulus and preceding white stimulus.

ordinate are given the variac settings, i.e. the brightness of the red stimulus at the point of "optimum green" in arbitrary units. On the abscissa are given the varying durations of the black interval separating the red flash and the preceding white flash. It will be seen that as predicted subjects under dexedrine have the lowest thresholds, subjects under amytal have the highest thresholds, while subjects under placebo are intermediate.

TABLE I

Source of Variation	d.f.	S.S.	V.Est.	V.R.	Significance
People	5	25438·6	5087·8	64·00	·01%
Days	2	1400·9	700·5	8·81	·01%
Treatments	2	3037·1	1518·6	19·10	·01%
Intervals	7	350·0	50·0	0·63	N.S.
Intervals/Treatments	14	692·1	49·4	0·62	N.S.
Intervals/People ..	35	3852·3	110·1	1·380	N.S.
Residual	78	6234·6	79·5	—	—
Total	143	41005·6			

Table I gives the numerical results of the experiments; it will be seen that the significant sources of variation, apart from the drugs, are people and days. The differences between people (individual differences) are not unexpected; the differences between days appear to be due almost entirely to the highly significant rise in the threshold from the second to the third day. Repetition of the experiment thus appears to work in the same direction as the administration of amytal.

T-tests were applied to the differences between the drugs, taken two at a time; these were all highly significant. The t-ratios are, respectively: Placebo vs. dexedrine—2·967; placebo vs. amytal—2·900; dexedrine vs. amytal—5·660. We may therefore conclude that the present experiment gives results very clearly in line with our hypothesis.

In the second experiment, a red flash of 20 msec. duration was followed by a variable length of white stimulation, from 40 msec. to 125 msec. The results of this experiment are shown in Figure 2; it will be seen that higher thresholds

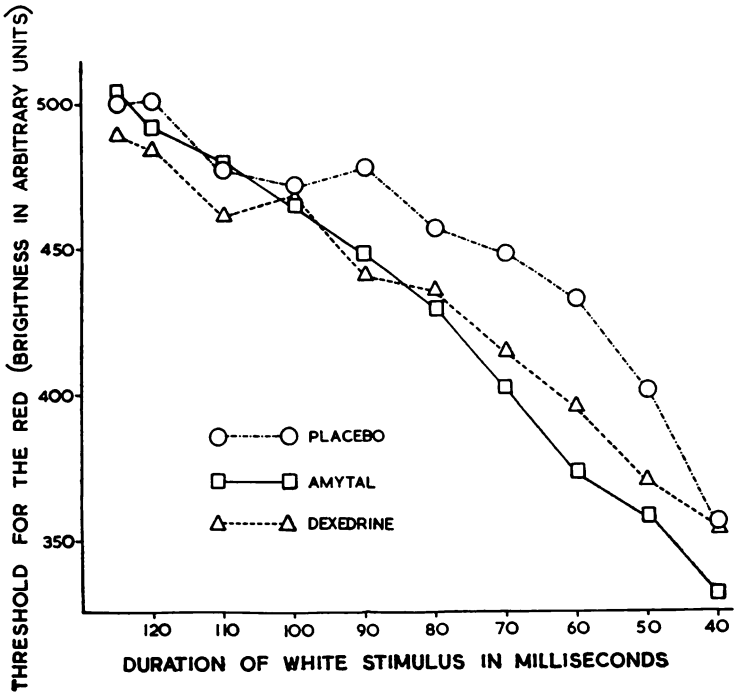

FIG. 2.—Threshold values for three drug groups plotted against duration of white stimulus following red stimulus.

are associated with longer durations of white regardless of drug used. Full results are given in Table II; all four primary sources of variation can be seen

TABLE II

Source of Variation	d.f.	S.S.	V.Est.	V.R.	Significance
People	5	7559·5	1511·9	39·48	·01%
Days	2	707·5	353·8	9·24	·01%
Treatments	2	554·2	277·1	7·23	·01%
Duration	9	11372·0	1263·5	32·99	·01%
Duration/Treatments	18	404·5	22·5	0·59	N.S.
Duration/People ..	45	1688·9	37·5	0·98	N.S.
Residual	98	3757·8	38·5	—	—
Total	179	26044·4	145·5	—	—

to have highly significant effects. Treatments, although the least effective, nevertheless give significant results. T-tests were applied to assess the significance of differences between pairs of drugs. The differences between dexedrine and amytal were not significant, but placebo produced higher thresholds than dexedrine ($t=3\cdot062$) or amytal ($t=3\cdot504$).

The result of this experiment is not in agreement with our hypothesis. Both dexedrine and amytal produced a lowering of the threshold, as compared with the placebo condition; this is correct for the dexedrine, but amytal had been expected to have the opposite effect. It will be noted that the effects which are contrary to expectation appear at low values of duration of the white flash; at 120 msec. the three drugs have sorted themselves into position as predicted. This is possibly important because in the first experiment we have shown that at 125 msec. the predicted effects occur at a high level of significance. Why the amytal should have paradoxical effects at low white flash duration values is not obvious to us; in so far as it fails to account for this effect the theory is obviously not yet complete.

SUMMARY

Two experiments were carried out to test predictions deriving from a general theory of drug action regarding the suppression of the primary visual stimulus. The first experiment supported the predictions at a high level of significance. The second experiment gave results with respect to placebo and stimulant drug which were in accordance with the theory, but the effects of amytal went counter to the theory. It was suggested that these contrary effects might be due to timing differences.

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