The Effect of Sedative Drugs on Human Performance

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INTRODUCTION

The aim of the experiments reported here was to measure the effect of outpatient doses of sedative drugs on human performance. Despite the large number of studies in this area there were no clear guidelines as to the appropriate techniques for measuring drug action on performance. Trouton and Eysenck concluded that this situation was caused by a lack of a sophisticated theoretical and methodological approach.20 Clayton pointed out that the diversity of techniques in psychopharmacology make it extremely difficult to reach any firm conclusions.4

The critical factor in this study was to isolate a sensitive performance parameter. Because of the anomalous nature of the area and the lack of strong theoretical guidelines it was not readily apparent how to select a performance task. However a number of papers have indicated that parameters of attention may be particularly sensitive to drug action. Evans and Davis concluded that doses of secobarbital sodium did not affect memory, but affected parameters of attention and the formation of higher order memorising strategies.5 Talland and Quarton indicated that pentobarbital affected strategies of information storage and recall.18 Hutt, Jackson, Belsham and Higgins found that phenobarbitone made subjects(Ss) capable of only short periods of attentiveness.11 Mirsky and Kornetsky indicated the sensitivity of a test of central attentional capacity and associative functions to sedative drug action.15

Hamilton and Copeman7 showed that doses of alcohol (0.17 and 0.55g of alcohol per 100 cc blood) produced attentional changes on a task devised by Bursill.3 Subjects were required to carry out a pursuit tracking task while simultaneously monitoring an arc of six lights positioned either side of it. This type of task has been used with a number of environmental stressors to determine their effects on selective attention.

Hockey looked at the effects of noise, an arousal stimulus, and sleep loss, a de-arousal stimulus, on selective attention. Using the Bursill equipment, where the tracking task was emphasised by instructions as being of primary importance, Hockey found that noise caused an increase in attentional selectivity.9 This was indicated when primary task performance was improved as well as the detections of centrally located light signals, but detection of peripheral signals was impaired. Another experiment looked at the effect of sleep loss on selective attention and determined that there was a monotonic relationship between the level of arousal and the degree of selectivity.10 Sleep loss caused centrally located light signals to be impaired as well as peripheral signals.

The aim of the present study was to determine: (a) whether a measure of selective attention was sensitive to outpatient doses of drugs which cause sedation; and (b) the effect of drug action on the structure of attention in a multi-component, complex, task. The task used was modelled on the Bursill equipment as used by Hamilton and Copeman.7

Royal Australian Navy Research Laboratory, Darlinghurst, N.S.W. Australia. This research was completed by the author while a Ph.D. student at Macquarie University. The work was subsidised by Riker Laboratoires, Australia Pty Ltd.
METHOD

Seven treatments were administered: a placebo, 2.5 and 5.0 mg of diphenylpryaline (an antihistamine), 50 and 100 mg of amylobarbitone (a barbiturate) 20 and 40 mg of chlorpromazine (a neuroleptic). Treatments appeared identical and were administered double blind.

Subjects

The four male subjects were paid volunteers from an introductory course in psychology. Each subject was passed suitable for the experiment by a medical practitioner. During the course of the experiment subjects were not allowed to take other pharmaceutical preparations. Coffee, tea and alcohol were not permitted to be consumed for twenty-four hours before test sessions. While no restriction was placed on smoking, subjects were asked to get a good night's sleep (7 to 8 hours) before test sessions. An interview was conducted when the subject arrived at the laboratory to see if these requirements had been carried out.

Performance Test

The apparatus is shown in Figure 1. Both tasks were incorporated in a 137 cm diameter translucent, white perspex hemisphere. The chair was adjustable in height so each subject could be positioned with his head at the centre of the hemisphere. All tasks were mounted on the equator, at eye level. The tracking task consisted of two edgewise meters mounted one on top of the other, directly in front of the subject. The top meter pointer was driven by a pseudo-random generator so that the pointer oscillated slowly across the meter. The pointer on the lower meter was controlled by the subject's manipulation of a single axis lever mounted on the right armrest of the chair. The task was to keep the bottom pointer within 2 mm either side of the top pointer. The score was taken as the length of time on target (TOT) over a three minute trial. Verbal feedback was given after each trial on TOT score. The peripheral detection task consisted of six light sources positioned at 20°, 50° and 80° either side of the tracking task and behind the translucent hemisphere. A signal consisted of an 0.5 cm diameter spot of white light which appeared on the inner surface of the sphere for 250 ms. The intensity of a signal could be varied by neutral density filters placed between the incandescent light bulb and the fibre optic which transmitted the light to the back of the hemisphere. Signal intensities were varied, during a pilot study, to give nominally 40, 60 and 80 per cent detections at the 80°, 50° and 20° locations respectively. Signal events occurred randomly across the six positions. A mean density of 12 events per minute were presented. A verbal response was given by subjects localising any signals they saw. Tasks were performed simultaneously and each measurement period lasted for fourteen consecutive three-minute trials.

Procedure

Two forty-minute training sessions were conducted to familiarise the subject with the performance test. In the experimental sessions the drugs and placebo were administered in a green 20cc solution which contained suitable taste-masking ingredients. The solutions for each treatment were labelled according to a code. The code was not revealed to the experimenter until after the data had been collected. The order of drug administration was randomised across subjects. Each subject attended seven, six-hour experimental sessions at weekly intervals at the same time of day. During the six-hour stay at the laboratory, the subject read or worked on university assignments. Measurement sessions, on the performance task, were carried out at one and five hours after drug ingestion.
RESULTS

Tracking Task

An analysis of variance (ANOVA) was conducted on these data and revealed a significant main effect of trials ($F = 3.27$, $df = 13/39$, $p < 0.01$) and a significant drug by trial interaction ($F = 1.67$, $df = 78/234$, $p < 0.05$). Tests for trend were conducted for the drug by trial by session interaction. Tests of linear, quadratic and cubic trends were conducted. Interpretation of higher trends was considered impractical.

First Testing Session. The 100 mg dose of amylobarbitone caused a significant negative linear trend over trials one to fourteen ($p < 0.05$). Both doses of chlorpromazine caused negative trends in the tracking data; 20 mg produced a linear trend and 40 mg produced a quadratic trend. The placebo condition produced a significant cubic trend.

Second Testing Session. The only treatments to produce significant results were the 100 mg dose of amylobarbitone and the 20 mg dose of chlorpromazine. Both produced significant negative linear trends over trials ($p < 0.05$).

Detection Task

To normalise peripheral detection data an inverse sign transformation (Johnson and Leone, 1964, p.56) was carried out. An ANOVA determined that there was a significant interaction between the angle of the peripheral event, and the side or quadrant in which the event occurred. Therefore data could not be summed over quadrants. Moreover while there was a significant effect of trials ($F = 8.47$, $df = 13/39$, $p < 0.01$) there was no drug by trial interaction ($F = 0.98$, $df = 78/234$, $p < 0.05$).
The experiment was primarily concerned with the effect of treatments on peripheral detection. Therefore mean comparisons of detection data for treatments were conducted for each light position and session. Three groups of pairwise comparisons were conducted where the family of comparisons was confined to both doses of the same drug and the placebo treatment. Critical values for these comparisons were computed using the Tukey (a) procedure. The variance estimate ($\sigma^2$) was taken as the pooled error variance for all treatments.

The results of these comparisons are shown in Figure 2. The placebo data are used as a baseline with drug treatment data being deviations from it. A boundary area shows the extent of the two tailed critical value ($p<0.05$). On these figures a positive value indicates impaired performance and a negative value improved performance.

#### Figure 2  Peripheral signal detection results.

* Drug treatment data are shown as deviations from placebo performance. The boundary lines denote the critical value ($p<0.05$) for significance testing.

In Figure 2, the results for diphenylpyraline showed tendency for the 2.5 mg dose to improve performance at the $80^\circ$ signal locations while impairing $20^\circ$ performance. The larger dose generally followed these trends but showed only significant impairment of performance across stimulus angles.

The results for amylobarbitone, also presented in Figure 2, showed that both doses impair performance across signal locations at the first testing session. At the second testing session there was a trend for $80^\circ$ signal location performance to improve while some $20^\circ$ and $50^\circ$ performances were impaired. This trend was most apparent for 50 mg dose(s).

The results for chlorpromazine, see Figure 2, showed that both doses significantly decrease performance over all stimulus positions. At the second session it was apparent that $20^\circ$ and $50^\circ$ signals tend to be impaired more than $80^\circ$ signals.
DISCUSSION

The results demonstrated that this performance task was sensitive to the effects of small doses of sedative drugs. The tracking performance data were generally consistent with the expectation of a decrease in performance over trials. However the cubic trend for the placebo treatment was anomalous. While this trend has both positive and negative components there was no indication of an overall decrease in performance over trials.

Attentional selectivity as indicated on the peripheral task demonstrated two levels of sedative action. At the first stage there was a broadening of attention to less important stimuli. This was shown by 80° signal performances being relatively better than the more central locations. At the second stage the degree of selectivity remained the same but the overall performance level decreased. This trend was exemplified by the detection scores for chlorpromazine being significantly impaired.

These findings support two general conclusions which are in agreement with Hockey’s findings. Firstly sedation causes a reduction in selectivity of attention. Secondly that sedation does not lead to unsystematic changes in selectivity. Relative performance appeared tied to some phenomenal priorities across the multicomponent task.

In order to refine these conclusions further a second experiment was conducted. This experiment manipulated arousal by using the stimulant caffeine and the sedative action of amylobarbitone, each at three dose levels. The peripheral detection task was modified so that parameters derived from signal detection theory (SDT) and information theory (IT) could be analysed.

An experiment by von Wright and Mikkonen used a similar task to derive SDT parameters for the effects of alcohol on performance. They were unable to compute SDT indices because of the extremely low false alarm rates for peripheral detection. To overcome this rather common finding for the Bursill task configuration, the signal positions were re-located at 25°, 30° and 35° either side of the tracking task. This change aimed at increasing confusion in stimulus identification. The location of the signals straddled the 30° boundary between the stationary eye field, where simple discrimination can take place without eye movement and the eye field, where eye movement is required for simple discrimination.

METHOD

Seven treatments were administered: Placebo; 75, 150 and 300 mg of caffeine; 50, 75 and 125 mg of amylobarbitone. The administration and double blind procedure was the same as that used in the previous experiment.

Subjects

Seven male, paid volunteers who came from an introductory course in psychology acted as subjects. They were medically screened for these drugs.

Performance Task

The tracking task was the same as for the previous experiment. The light sources for the detection task were positioned at 25°, 30° and 35° either side of the tracking display. The probability of detection for an event was maintained at 40, 60 and 80 per cent for the 35°, 30° and 25° location respectively. Each trial lasted 400 seconds and contained 100 decision units (DUs). Ninety-eight of the DUs were used to present one of seven events. The events were either a 250 ms flash from one of the six stimulus locations or no stimulus at all. Each
of these seven events occurred fourteen times in each trial. The remaining two DUs at the end of the trial were used for feedback on tracking performance. Each testing session lasted for five consecutive trials. The order of peripheral events was randomised across DUs and trials with the restriction that no single event could occur consecutively. After an event had been presented a 50 dB A audio signal was given. This indicated that the subject had to make a verbal response by either localising the stimulus or indicating that no stimulus occurred. Verbal responses were recorded on a 7 by 7 matrix where the stimulus presented formed one axis and the response given, the other.

**Procedure**

The procedure followed that of the previous experiment.

**RESULTS**

**Tracking Task**

An ANOVA revealed a significant trend over trials for central tracking performance \((F = 22.39, df = 4/24, p<0.01)\) but no drug by trial interaction \((F = 1.15, df = 24/144, p>0.05)\). Pairwise comparisons were carried out using the Tukey (a) procedure between means summed over trials. Two families of comparisons were made, one between placebo and caffeine doses and the other between placebo and amylobarbitone.

These comparisons revealed that 300 mg of caffeine significantly \((p<0.05)\) improved tracking performance at the first testing session and 125 mg of amylobarbitone decreased performance over both testing sessions.

**Peripheral Detection Task**

An ANOVA on the correct detection data revealed that there was a significant effect of trials \((F = 9.17, df = 4/24, p<0.01)\), but no drug by trial interaction \((F = 0.85, df = 24/144, p>0.05)\). All further analyses were based on data summed over trials.

*Probability of Detection (Pr D).* An ANOVA revealed that there was no interaction between stimulus angle and the quadrant in which the event occurred \((F = 3.48, df = 2/12, p>0.05)\). Therefore data were summed over quadrants. Two families of pairwise comparisons were made, using the Tukey (a) method. Comparisons were made between doses of caffeine and placebo and doses of amylobarbitone and placebo. Significant \((p<0.05)\) results were found for the largest doses of each drug. The data are presented in Figure 3. The Pr D was significantly improved for the 25° stimulus position after 300 mg of caffeine at the first testing session only. Performance was impaired at the 30° and 35° positions after 125 mg of amylobarbitone also at the first session only.

*Probability of False Alarm (Pr FA).* This parameter was calculated, however no significant \((p<0.05)\) drug effects were found.

*Sensitivity P(I).* The non parametric index of sensitivity\(^6\) showed that 125 mg of amylobarbitone decreased P(I) to the 30° and 35° stimuli at the first testing session. These were the only significant contrasts. Analyses of Pr D, Pr FA and P(I) for events where no light signal was presented did not reveal any significant \((p<0.05)\) contrasts.
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KEY:

PLACEBO

Δ ↔ Δ CAFFEINE 75mg

○ ↔ ○ CAFFEINE 150mg

× ↔ × CAFFEINE 300mg

PLACEBO

Δ ↔ Δ AMYLOBARBITONE 50mg

○ ↔ ○ AMYLOBARBITONE 75mg

× ↔ × AMYLOBARBITONE 125mg

SESSION 1

SESSION 1

Figure 3  Peripheral probability of detection (PrD) results.

Drug treatment data are shown as deviations from placebo performance. The boundary lines denote the critical value (p<0.05) for significance testing.

Information Theory Parameters

Three parameters from IT were calculated based on the formulations of Attneave. Data for each parameter were calculated for each drug treatment and testing session. The three parameters were: \( H_y(x) \), which indicated the component of stimulus information lost by the subjects; \( T(x;y) \), the amount of information transmitted through the subjects; \( H_x(y) \), which represented the noise component in the response. Mean comparisons (Tukey (a) method) across treatments and sessions showed that 125 mg of amylobarbitone impaired performance over all IT parameters compared to placebo and 75 mg of the same drug at the first testing session. No other contrasts were significant.

DISCUSSION

In summary there were three major findings from the second experiment. Firstly, the improvement of tracking and 25° Pr D performance for caffeine. These results were in agreement with the hypothesis that arousal increases attentional selectivity. Secondly, the impairment of tracking and 30° and 35° Pr D performance for amylobarbitone was contrary to the hypothesis that decreased arousal should cause a loss of selectivity. Thirdly, only the largest dose of each drug produced significant performance changes. This result was contrary to the findings of the first experiment which showed the task sensitive to the lower amylobarbitone doses administered.

The result for amylobarbitone and the lack of sensitivity of the performance task may be explained in terms of an interaction between arousal levels and task complexity.

Hockey argued that the level of task complexity, defined by signal rate and number of sources, interacted with arousal levels which were modified by noise stimuli. Teichner proposed a model of information processing based on task complexity and arousal levels.
The model predicts that decreased arousal broadens the attentional filter. The bandwidth of this filter was tuned to maximise acceptance of task relevant signals into a short term memory store. However the filter was also affected by the bandwidth of the desirable incoming signal and the arousal level. The model predicts that decreased arousal causes an increase in filter bandwidth which was consistent with findings that decreased arousal causes a decrease in attentional selectivity, that is an increase in the probability of reacting to task irrelevant signals. Such a model matches the data for the first experiment and Hockey's conclusions. But Teichner's model goes on to predict that if the signal input rate exceeds the capacity of higher cognitive processes to sample and respond, then the filter bandwidth is reduced, that is an increase in selectivity occurs. This reversal in selectivity prevents the cognitive system from being overloaded.

The results for 125 mg of amylobarbitone indicated an increase in attentional selectivity by 25° Pr D performance being maintained at the expense of 30° and 35° performance. While these findings were contrary to the results of the first experiment and Hockey's data they were exactly the same as findings reported by Hamilton and Copeman. They found that alcohol, clinically a depressant at the doses used, decreased tracking performance and detection on extreme peripheral signal locations but central signal location performance was not changed.

A change in the task parameters between the first and second experiments, leading to increased task complexity, may have caused a reversal in selectivity to occur as predicted by Teichner's model. In the first experiment 12 peripheral signals occurred every minute. The average response rate was between five and ten subject paced responses per minute, depending on how many peripheral signals were detected. In the second experiment the responses were experimenter paced and increased to 15 per minute. The increased number of experimenter paced responses may also have caused response induced stimulation. Adams, Stenson and Humes found that increased frequency or complexity of responses had a stimulant effect which improved performance on vigilance and attention tasks. This task induced stimulation may have masked the sedative effects of 50 and 75 mg of amylobarbitone in the second experiment. An extension to this hypothesis would predict a potentiation of caffeine's action. The data showed no improvement of performance for the 75 and 150 mg caffeine doses. However the validity of this proposal cannot be supported as there was no data for task sensitivity for caffeine using the parameters of experiment one.

The failure of the SDT and IT parameters to yield any worthwhile data may be attributed to the high response rate. Moray concluded that the SDT approach was insensitive to mechanisms of attention because of the rapid presentation of events and the high proportion of targets contained in them. Jerison proposed that SDT parameters do not provide information about the way signals are received (attention), but deal with how the received signal is processed.

It was therefore concluded that a simple model of attentional selectivity bases on arousal levels is inappropriate for evaluation of drug effects on performance. A more appropriate model should include parameters of task complexity.

REFERENCES
