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16. Abstract

Thirty young men were randomly assigned in equal numbers to one of the following groups: placebo (lactose), secobarbital (100 mg), or d-amphetamine (10 mg). The drugs or placebo were administered in capsules in a double-blind procedure following practice at a tracking task and baseline determinations of tracking performance levels in both static (stationary) and dynamic (angular acceleration) conditions. Tests were scheduled 1, 2, and 4 hours after capsule ingestion; all tests were conducted inside a Stille-Werner rotator and were in total darkness with the exception of the illuminated tracking display. With the rotator stationary, d-amphetamine subjects performed significantly better than controls during the 2-hour and 4-hour post-drug sessions; no other static differences among the groups were significant. However, during angular acceleration, secobarbital subjects made significantly more tracking errors and had significantly more vestibular nystagmus than both the control and the d-amphetamine groups for all post-drug sessions. These findings agree with our previous studies of alcohol effects: depressant drugs may have little or no deleterious influence on tracking performance in static environments, but may produce marked performance degradation during angular motion. The primary cause of this performance impairment appears to be a vestibulo-ocular one; the ability to inhibit vestibular nystagmus by visual fixation is impaired.

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EFFECTS OF SECOBARBITAL AND D–AMPHETAMINE ON TRACKING PERFORMANCE DURING ANGULAR ACCELERATION

I. Introduction.

Most studies of the effects of drugs on performance have been conducted under stationary conditions. In an aviation environment, however, an important aspect of drugs concerns their effects on performance during motion. Recent joint studies by CAMI and USNAMRL[3 6 12] have shown that the deleterious effects of alcohol on psychomotor performance in the laboratory are more pronounced during angular motion than under stationary conditions. This difference may be attributed to the interfering effects of alcohol on the ability of the subject to reduce with visual cues the nystagmic eye movements which occur as a result of angular stimulation of the vestibular system. Since alcohol acts primarily as a central nervous system depressant, it seems likely that other drugs which have depressive effects may similarly affect an individual's visual fixation ability and, consequently, his psychomotor performance during vestibular stimulation. Moreover, there is no information presently available concerning the effects of an analeptic, as opposed to a depressant, on eye-hand coordination during concomitant vestibular stimulation. Consequently, the purpose of this study was to extend our knowledge of the interaction of tracking performance, angular acceleration, and drugs to include the effects of both a commonly used depressant other than alcohol (secobarbital) and a commonly used analeptic (d-amphetamine).

II. Method.

Subjects. Thirty male college students ranging in age from 20 to 30 years served as subjects. Before any testing, 10 subjects each were randomly assigned by a double-blind procedure to one of three groups: a placebo, secobarital, or d-amphetamine group. None had any previous laboratory experience involving vestibular stimulation. Subjects were not allowed to smoke or drink beverages containing caffine, except during the 2-hour lunch period which preceded the final test session.

Apparatus. The angular stimulation was provided by a modified Stille-Werner rotation device. The subject was seated in an upright position directly over the center of rotation, with his head fixed in a headrest so that the lateral semicircular canals were approximately in the plane of rotation. The device was programmed, using a Wavetek signal generator, to provide a triangular waveform stimulus of 10°/sec^2 with a period of 48 seconds and a peak velocity of 120°/sec in both clockwise and counterclockwise directions.

A one-degree-of-freedom compensatory tracking task consisting of an aircraft localizer/glideslope indicator (attached to the front of the rotation device and a joy stick (mounted directly in front of the subject) was used as a measure of psychomotor (eye-hand) coordination. The vertical needle on the instrument was deflected to the left and right of center by a sinusoidal forcing function with a 14-second period. The needle movement was thus in the same approximate plane as the eye movements arising from the vestibular stimulation. The subject's task was one of keeping the needle in the center or null position by compensatory movements of the joy stick. Deviations from the null position were considered as errors and a voltage proportional to these deviations was electronically integrated over 1-second intervals and recorded. Further details concerning the operation of the tracking task are presented elsewhere.[5]

Light was projected through a tube to illuminate the display. A card, coated with the same white paint as the needle, was placed in front of the instrument for luminance calibrations.

We gratefully acknowledge assistance in data collection and scoring by Carolyn Eubanks, Martha Manley, and Cynthia Mitchell.

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The luminance was checked with a MacBeth illuminometer and set at one ft.L. The test room was otherwise in total darkness.

Electrodes were taped beside the outer canthus of each eye to record horizontal eye movements. Calibration of the horizontal eye movements was accomplished by having the subject sweep his eyes between two small flashing lights on the front of the rotator; the lights subtended a visual angle of 15°. Eye movements and tracking errors were recorded on a Beckman Type T electroencephalograph.

Procedure. Following instructions, each subject was given two minutes of static tracking practice, i.e., with the rotator stationary. This was followed by a practice session comprising one minute of static tracking, 2.5 minutes of dynamic tracking (during five complete cycles of rotation), and three minutes of rotation in the dark (the latter was conducted for purposes other than those of the presently reported experiment). A pre-drug session and three post-drug sessions were identical to the practice session.

Following the pre-drug session, the subjects consumed their respective capsules on an empty stomach: either 10 mg of d-amphetamine, 100 mg of secobarbital, or a placebo containing lactose. The drugs were administered using a double-blind procedure. Post-drug sessions were conducted 1, 2, and 4 hours after administration of the drugs with blood pressure and heart rate monitored immediately prior to each of the test sessions.

Scoring. One-second intervals of tracking error were measured, summed, and averages obtained for both the static and dynamic conditions. The amount of slow-phase eye displacement was measured and the number of nystagmic eye movements was counted across the last two cycles of rotation during dynamic tracking. Mean values in degrees per second and beats per second were calculated and used as measures of nystagmic output. Scoring was accomplished without knowledge of the group (drugs or placebo) to which any subject belonged, and these absolute values were used in the statistical analyses.

Table 1

Mean pulse rates and blood pressure measures (systolic/diastolic) obtained during each pre- and post-drug session for subjects in the secobarbital, d-amphetamine, and placebo groups.

		Post-Drug Sessions		
Group	Pre-Drug	1-hr.	2-hr.	4-hr.
		Pulse Rate		
secobarbital	65.1	65.2	65.5	70.0
d-amphetamine	66.7	66.0	70.9	78.2
placebo	72.2	65.8	67.2	72.6
		Blood Pressure		
secobarbital	115/73	114/73	112/73	117/72
d-amphetamine	116/73	122/75	126/76	111/73
placebo	116/77	120/79	117/79	121/77

Table 2

Levels of statistical significance (Not Significant; $p < .05$; $p < .01$; $p < .001$) obtained within groups by t tests. Comparisons were made between tracking scores and measures of nystagmus from the pre-drug session and each post-drug session for the secobarbital d-amphetamine, and placebo groups separately.

		Pre-Drug Session vs.		
Measure	Group	1-hr.	2-hr.	4-hr.
Static Tracking Error	secobarbital	N.S.	N.S.	N.S.
	d-amphetamine	N.S.	.05*	.01*
	placebo	N.S.	N.S.	N.S.
Dynamic Tracking Error	secobarbital	.01**	.001**	N.S.
	d-amphetamine	N.S.	.001*	.05*
	placebo	N.S.	N.S.	.01*
Slow-Phase Nystagmus	secobarbital	.01**	.01**	.01**
	d-amphetamine	N.S.	.01*	.001*
	placebo	.05*	N.S.	N.S.
Nystagmic Beats	secobarbital	.001**	.001**	.01**
	d-amphetamine	N.S.	.05*	N.S.
	placebo	N.S.	N.S.	N.S.

* indicates significantly less of the measure
** indicates significantly more of the measure

For graphic presentation, however, "change" scores were computed. For each group and each measure, the mean score for the pre-drug session was plotted as a zero base and the percentage of increase or decrease in subsequent scores for a given measure (i.e., in sessions which followed administration of a drug or placebo) was plotted as "per cent increase" or "per cent decrease" from the pre-drug level.

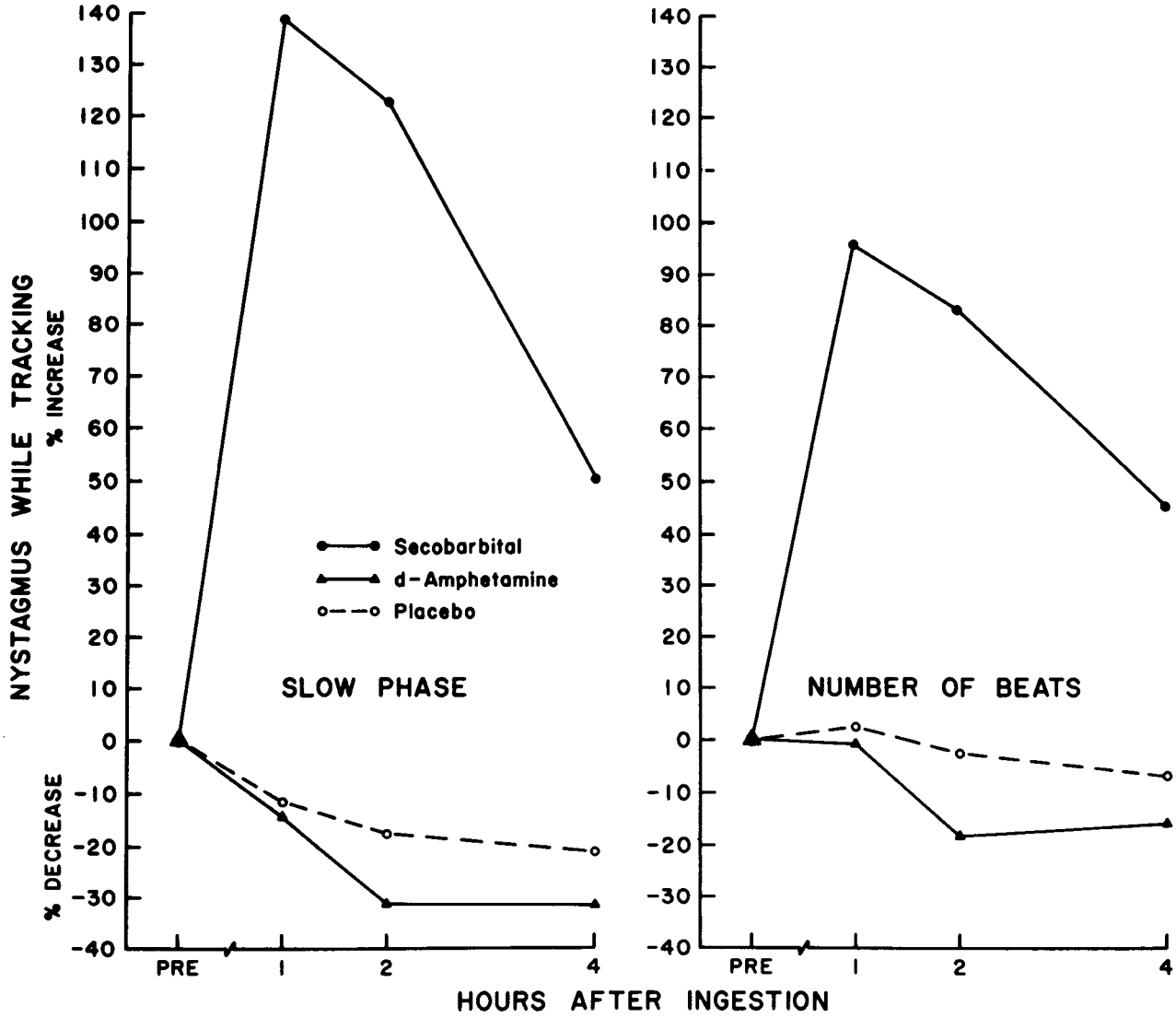

FIGURE 1. Changes in tracking performance under static (stationary) and dynamic (angular acceleration) conditions. Error scores for the pre-trial (before ingestion of the drug or placebo capsule) were set at 0. Error scores for the post-ingestion sessions were converted to percentages of increase or decrease from the pre-ingestion baseline.

III. Results.

Cardiovascular Measures

Means and standard deviations for heart rate and blood pressure are presented in Table 1. A rise in both measures occurred for the d-amphetamine group after administration of the drug, but these changes were not statistically significant at the .05 level. Moreover, none of the measures of heart rate or blood pressure for either the secobarbital or control group changed significantly ($p>.05$) across sessions. (Goldstein, Searle, and Schimke[7] also failed to find any effect of 10 mg of d-amphetamine or of 200 mg of secobarital on blood pressure and heart rate, although Collins and Poe[4] reported statistically reliable increases in these measures following 12 mg doses of amphetamine administered subcutaneously.) Thus, the presence or absence of the drugs could not be ascertained by these cardiovascular measures.

Static Tracking

Within-Group Comparisons of Tracking Performance. Subjects in the placebo and the secobarbital groups evidenced little change in static tracking error from the pre-drug through the

Table 3

Levels of statistical significance (Not Significant; $p < .05$; $p < .01$) obtained between groups with analyses of covariance of tracking and nystagmus scores. Duncan's New Multiple Range Test was then used to determine which of the secobarbital (S), d-amphetamine (D), and placebo (P) groups differed (< ; >) in scores during the sessions following drug administration.

	Post-Drug Sessions		
Measure	1-hr.	2-hr.	4-hr.
Static Tracking Error	N.S.	.05 D < P	.05 D < P
Dynamic Tracking Error	.01 S > D S > P	.01 S > D S > P	.01 S > D S > P
Slow-Phase Nystagmus	.01 S > D S > P	.01 S > D S > P	.01 S > D S > P
Nystagmic Beats	.01 S > D S > P	.01 S > D S > P	.01 S > D S > P

post-drug sessions (Figure 1); all of the post-drug static tracking error means were within ±11% of their respective pre-drug levels and none of these differences was statistically significant at the .05 level by *t* test (Table 2). In contrast, subjects in the d-amphetamine group evidenced a steady decrease in tracking error from the pre-drug session through the 4-hour post-drug tests. Some improvement with practice in static tracking performance at this task is not an unusual finding even for non-drugged subjects.[3,6] However, while most of the improvement in tracking performance for the d-amphetamine group occurred during the session conducted 1

hour after drug administration (18%), only the 2-hour and the 4-hour post-drug scores were statistically lower than the pre-drug level ($p<.05$ and .01, respectively).

Between-Group Comparisons of Tracking Performance. Comparisons of static tracking performance among the three groups were made using analysis of covariance and Duncan's New Multiple Range Test; results are presented in Table 3. During static tracking, the only statistically significant differences ($p<.05$ in both cases) occurred between the placebo and d-amphetamine groups for the 2-hour and 4-hour post-drug sessions; the d-amphetamine group performed with less error during these sessions. Thus, d-amphetamine had some positive effect on static tracking performance; a stronger conclusion does not seem warranted since (a) the placebo group in this study showed little performance improvement while in two previous studies[3,6] control groups decreased tracking errors by approximately 15% over similar time and test periods, and (b) there were no significant differences in performance between the group receiving d-amphetamine and the secobarbital group.

Dynamic Tracking

Within-Group Comparisons of Tracking Performance. Both the d-amphetamine and control groups exhibited steady improvement in dynamic tracking performance across sessions. This pre-drug to post-drug improvement for the two groups (which must be considered largely as a practice effect[3,6,12]) ranged from 3% to 28% across sessions (see Figure 1) and was statistically significant by t tests two hours and four hours after administration of the capsules ($p<.001$ and .05, respectively) for the d-amphetamine group, and four hours post-administration ($p<.01$) for the placebo group (Table 2). Although there was an initial tendency for the subjects receiving the d-amphetamine to show slightly greater performance improvement than the control subjects, none of the differences between the two groups was statistically significant. On the other hand, subjects in the secobarbital group evidenced a dramatic increase in tracking error (66%) one hour after receiving the drug; during subsequent sessions their tracking error was reduced, but the error scores four hours after administration of the drug were still 11% above the pre-drug levels. This pre-drug to post-drug deterioration in tracking performance for the secobarital group was statistically significant for the 1- and 2-hour post-drug sessions ($p<.01$ and .001, respectively).

Between-Group Comparisons of Tracking Performance. Results of an analysis of covariance and Duncan's New Multiple Range Test indicated that the dynamic tracking performance of subjects in the secobarbital group was significantly poorer than that of subjects in both the placebo and d-amphetamine groups for all of the post-drug sessions ($p<.01$ in each case). Although the d-amphetamine group had less tracking error than placebo subjects during the 1-hour and 2-hour post-drug sessions (and particularly during the latter when a maximal effect of the drug might be expected; see Figure 2), none of these differences was statistically significant.

Nystagmus During Tracking

Within-Group Comparisons of Nystagmic Output. With regard to both the slow-phase velocity and the number of nystagmic eye movements, subjects in the placebo and d-amphetamine groups exhibited a steady decline from the pre-drug through the post-drug sessions (Figure 2); several of these differences were statistically significant (Table 2). In contrast, for subjects in the secobarbital group, both slow-phase nystagmus and number of nystagmic beats increased significantly during the 1-hour post-drug session ($p<.01$ and .001, respectively) and, although there was a gradual return toward the pre-drug level, both measures of nystagmus for this group remained significantly above the pre-drug levels through the 4-hour post-drug session ($p<.01$).

Between-Group Comparisons of Nystagmic Output. The slow-phase velocity measures and the number of nystagmic beats recorded for subjects in each group were submitted to analyses of covariance and Duncan's New Multiple Range Test. Scores for both measures were significantly higher ($p<.01$ in every case) for secobarbital subjects than scores for the placebo and d-amphetamine groups during each of the post-drug testing sessions (Table 3). Only one appreciable difference occurred between the placebo and d-amphetamine groups (at the 2-hour post-drug sessions; see Figure 2) but that difference was not statistically reliable.

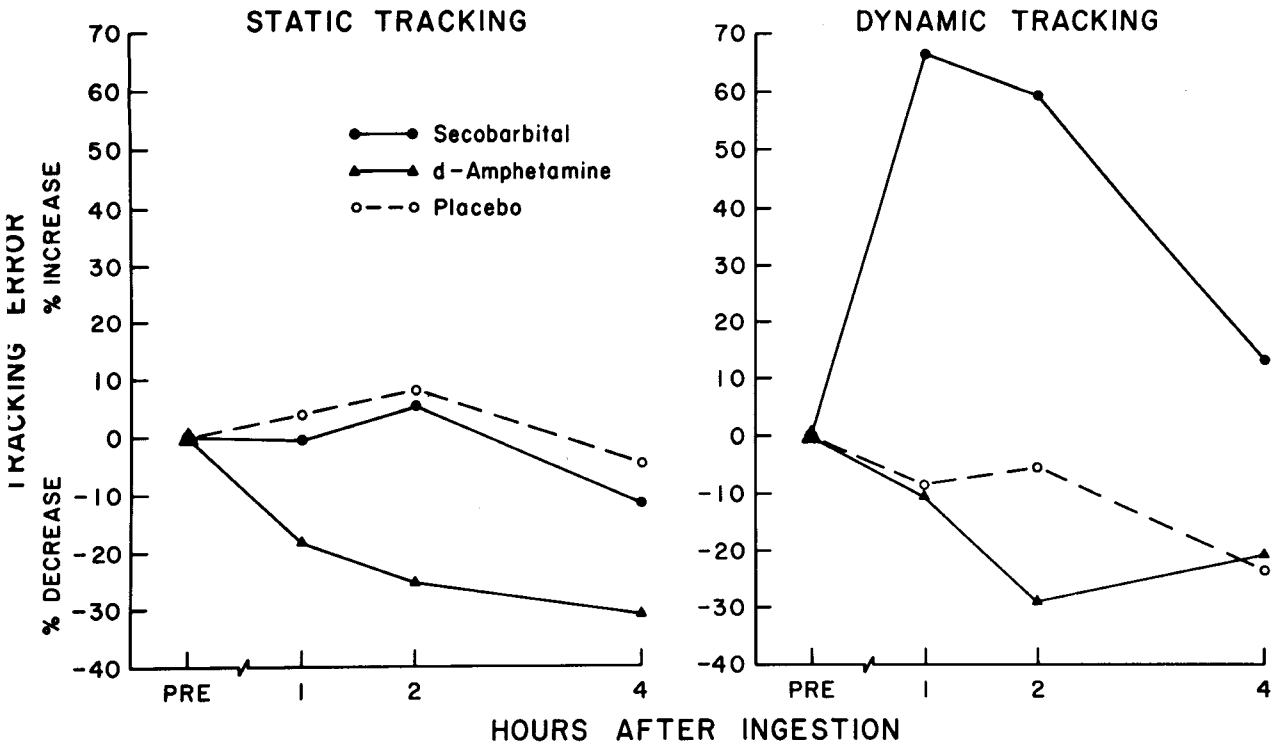

FIGURE 2. Slow-phase displacement of nystagmus and the number of nystagmic beats obtained while subjects were tracking in the dynamic condition. Measures for the pre-trial (before ingestion of the drug or placebo capsule) were set at 0. Measures for the post-ingestion sessions were converted to percentages of increase or decrease from the pre-ingestion baseline.

Conclusion

The obtained decrement in tracking performance for the secobarbital subjects during angular stimulation is correlated with a considerable increase in the number and velocity of nystagmic eye movements with attendant visual blurring. Thus, these deleterious effects of secobarbital appear to be similar to those produced by alcohol; both apparently interfere with the ability of the visual system to suppress nystagmic eye movements which result from angular stimulation of the vestibular system. Further support of such an effect of barbiturates appears in an earlier study of optokinetic and caloric nystagmus by Rashbass and Russell[10] where the authors found that the barbiturate sodium amytal "... abolished the inhibiting effect of visual fixation" (p. 355). Barbiturates apparently suppress optokinetic nystagmus[1 2] and interfere with smooth tracking movement[9 10] and visual fixation ability[10] in much the same fashion as does alcohol.[3 6 11 12]

The present data support our previous findings[3 6 12] with alcohol, i.e., the effect of at least some depressant drugs on performance may not be evident in a stationary environment, but may be substantial during angular stimulation of the vestibular system. Such degradation in performance appears primarily attributable to impaired visual acuity resulting from the inability to suppress vestibular nystagmus by visual means.[5 8] In addition to nystagmus, however, motion may contribute to performance declines by producing other effects, such as distraction, which essentially make the tracking task more complex and more susceptible to the influence of depressant drugs. d-Amphetamine, an alerting drug, appeared not to affect dynamic tracking although it had some facilitory effect on static tracking. In any event, future evaluations of effects of drugs on performance should take into account the influence of motion. Any drug which interferes with the ability of a pilot to fixate and read cockpit instruments during flight would severely compromise his ability to react properly during maneuvers, especially under IFR conditions.

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