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16. Abstract
Few data are available concerning the effects of sleep loss on vestibular responses although those responses are significant products of motion in aviation environments. This study assessed periodically throughout a period of approximately 55 hrs. of sleep loss the ocular nystagmus and motion experiences of men exposed to both simple (angular acceleration) and complex (Coriolis) vestibular stimulation. The effects on those reponses of an alerting drug administered after 54 hr of sleep loss were also examined. Control and sleep-deprived groups each comprised 10 young men. Angular accelerations and Coriolis stimulation (30-deg head movements during CW rotation) were accomplished in an enclosed Stille-Werner rotating device. Nystagmus and motion experiences (turning, "diving," and "climbing") were recorded throughout each session. Tests were given at 0900 and 1300 on each of 3 successive days. Subjects ingested 10-mg of d-amphetamine at 1200 on Day 3. During simple stimulation, the sleep-deprived group showed regular declines across sessions in slow phase and duration measures of nystagmus but fast phase ocular frequency and measures of experienced turning resisted declines until the final predrug session; response latencies increased with sleep loss. Declines during rotation for ocular output and measures of perceived displacement during rightward head tilts ("climbing" sensation) were obtained for the sleep deprived, but both nystagmus and sensations were unaffected by return (leftward) movements of the head ("diving" sensation). d-Amphetamine had no consistent effect on either the ocular or subjective responses of control subjects, but significantly increased nystagmus and elevated (but not significantly) measures of turning experiences for the sleep deprived.

17. Key Words
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EFFECTS OF SLEEP LOSS ON VESTIBULAR RESPONSES DURING SIMPLE AND COMPLEX VESTIBULAR STIMULATION

The known effects of sleep loss on vestibular responses are meager. Wolfe and Brown[1,2] gave each of 16 subjects two trials (one 8 deg/sec^2 acceleration and one 24 deg/sec^2 deceleration) before and after 24 hrs of sleep loss. Of four measures of nystagmus (slow-phase, frequency, duration, threshold) only the frequency counts for the 24 deg/sec^2 stimulus differed significantly from pre- to posttest; frequency was higher during the posttest, but the absence of a control group makes interpretation difficult. Dowd, Moore, and Cramer[3] reported no difference in the occurrence of motion sickness between rested and sleep deprived (24-hr) military pilots (N=131) during laboratory Coriolis stimulation, but differences in decay rates of vertical nystagmus and in "sensitivity" (a product of maximum eye velocity and decay rate of vertical components of nystagmus) were obtained. The design of the study, however, was such that clear attribution of the effects to sleep loss was not possible and results were presented from only one direction of head movement (that yielding a sensation of a "rolling climb"). Dowd[4] tested an additional group of 38 pilots and apparently included 105 subjects from the study noted above (the 26 pilots who had shown motion sickness symptoms were excluded) yielding a total of 143 experienced pilots divided into three groups (rest/rest, rest/sleep deprived, sleep deprived/rest); he reported a significant increase in the sensitivity measure and a significant decrease in the decay rate of vertical nystagmus (from a single head movement) following 24 hrs of sleep loss. He interpreted that increase as a weakening of vestibular suppression acquired through flying activities.

While nystagmus measures are important manifestations of vestibular function, the sensations of motion experienced during simple angular accelerations and Coriolis-type (complex) stimulation are especially significant in aviation environments. The present study was designed to assess the effects of sleep loss on a number of vestibular and vestibular-related responses including nystagmus and sensations over a longer period of sleep deprivation (approximately 55 hr) than previously investigated.

METHOD

Subjects. A control group and a group of sleep-deprived subjects each comprised 10 men, paid volunteers, 21-28 years old (mean age 23.3 years). All subjects remained under constant monitoring in the laboratory for the three experimental days. Half the subjects were allowed to sleep; the other half were kept awake and active. Subjects were asked to abstain from alcoholic drinks for 48 hours prior to the study, to arrange to have 8 or more hours of sleep on the night prior to the first experimental day, and to rise at 0700. Subjects were not allowed to consume caffeine drinks or to smoke throughout the study.

The assistance of Gregory N. Constant, Patricia Gant, Linda Foreman, Cissy Lennon, J. M. Lentz, and RuthAnn Parvin in the conduct of this study and of Deborah K. Taylor for aid in data analysis is gratefully acknowledged.

Procedure. Angular accelerations and Coriolis stimulation were accomplished in total darkness in an enclosed Stille-Werner rotating device. The subject's head was fixed in a head-holder; a bite-block helped to position the horizontal semicircular canals approximately in the plane of rotation.[1] The room was in total darkness and the head-holder was adjusted to permit uniform head tilts of 30 deg to the right and a return movement (30 deg to the left) to an erect head position. Subjects were instructed to keep their eyes open during trials and were periodically reminded to do so prior to each angular acceleration and each instruction to move their heads.

Each subject was accelerated clockwise at an angular rate of 5 deg/sec^2 for 18 sec to a constant velocity of 90 deg/sec; during this time he signaled his turning sensations. The signals were depressions of a microswitch to indicate when the subject experienced the start, successive 90 deg angles of turning, and the end of his turning sensations.

After approximately 3 min of constant rotation and following the cessation of the turning sensations, the subject was instructed to tilt his head to the right and, later, to return his head to an erect position. Each head tilt required about 1 sec and a rest period of at least 1 min followed the end of the response to each tilt and each return movement. The subject used the microswitch to signal the start and end of each "climbing" (head tilt) or "diving" (head return) sensation and then provided a verbal estimate of the number of deg of "climb" or "dive" he experienced. The chair was decelerated to a stop at 5 deg/sec^2 with the subject, head upright, signaling his turning experiences.

One day prior to the experimental sessions, subjects were given instructions and then participated in a set of familiarization trials to acquaint them with forms of stimulation produced by a laboratory rotator and to give them experience in signalling and rating their experiences of motion. This was followed by a formal practice session during which performance on each test was recorded.

Experimental tests were given at 0900 and 1300 on each of the 3 successive experimental days. Each subject ingested a 10-mg capsule of d-amphetamine sulphate at 1200 on Day 3.

Scoring

Turning Experiences. The turning motions experienced by both groups of subjects were examined in three ways. Calculations were made of (i) the latency of the response in sec (the time from the start of physical turning to the first signal of experienced turning), (ii) the duration of perceived turning in sec (the time from the first signal of experienced turning to the signal indicating the end of experienced turning), and (iii) the amount of experienced turning in deg (90 deg times the total number of turning signals minus 2--the two omitted signals being the indications of the start of turning and the end of turning).

Coriolis Sensations. Two measures of Coriolis sensations were obtained. The first was duration in sec of the climbing or diving experience resulting from head movements during rotation. Subjects depressed a microswitch when the sensation began and when it ended. The other measure was magnitude in deg of perceived climb or dive. Subjects estimated the amount of perceived motion in

the vertical plane; i.e., they estimated their peak apparent displacement in deg above ("climb") or below ("dive") an earth-horizontal plane.

Vestibular Nystagmus. Three measures of the horizontal components of nystagmus occasioned by angular accelerations and decelerations were obtained from the tracings, viz, slow-phase displacement, number of fast phases, and duration of response. The same measures were obtained for vertical components of nystagmus occasioned by head movements during rotation. Conversion of slow-phase measures into deg of eye movement was accomplished by means of calibrations obtained with two sets each of two small alternately flashing lights, subtending a visual angle of 15 deg in the horizontal plane in one case, and 15 deg in the vertical plane in the other case.

RESULTS

Statistical Analyses. In all cases, data for a given condition/measure (e.g., acceleration/nystagmus duration) were analyzed first by multivariate analysis (MANOVA) techniques for repeated measures and then by t tests in making paired comparisons. Separate sets of analyses (tables 1 and 2) were accomplished for (i) the first session (Day 1, 0900), the fifth session (Day 3, 0900), and the postdrug session (Day 3, 1300) to isolate the effects of conditions (i.e., sleep loss and drug), and (ii) for the first five sessions (Day 1, 0900 through Day 3, 0900) to clarify successive effects. The second set of analyses (see tables 3, 4, and 5) is occasionally noted in the text as an aid to interpreting findings from the main analyses.

Rotation-Induced Nystagmus. For the control group, nystagmus measures stayed relatively constant or showed a shallow decline across sessions (see figure 1). Results of the MANOVAs (table 1) and the paired comparison tests (table 2) indicated that there was no significant change in nystagmus output for the fifth vs. the first session to either acceleration or deceleration stimuli (in fact, there were no significant differences between any pairings of the first five sesions for any nystagmic measure; see tables 3 and 4). Ingestion of d-amphetamine by control subjects had minimal effect, viz. for decelerations only, it increased the duration measure for the postdrug session significantly above that of session 5 and increased the fast phase frequency score for the postdrug session significantly above that of the first session ($p < .05$ in both cases).

For sleep-deprived subjects, all measures of nystagmus during session 5 were below ($p < .01$) those of the first session for both acceleration and deceleration. Thus, sleep deprivation produced an overall decline in output of nystagmus (see table 2). The ingestion of d-amphetamine uniformly increased ocular output; that increase was significant in comparisons of the postdrug session vs. session 5 for all measures of nystagmus during acceleration stimuli, and for the fast phase frequency of nystagmus during decelerations. These significant increases elevated the postdrug scores sufficiently that they did not differ from the scores obtained in the first session. Where the postdrug increases were not significantly above session 5 (i.e., for duration and slow phase measures during deceleration), the output of nystagmus remained significantly ($p < .01$) below the first session measures.

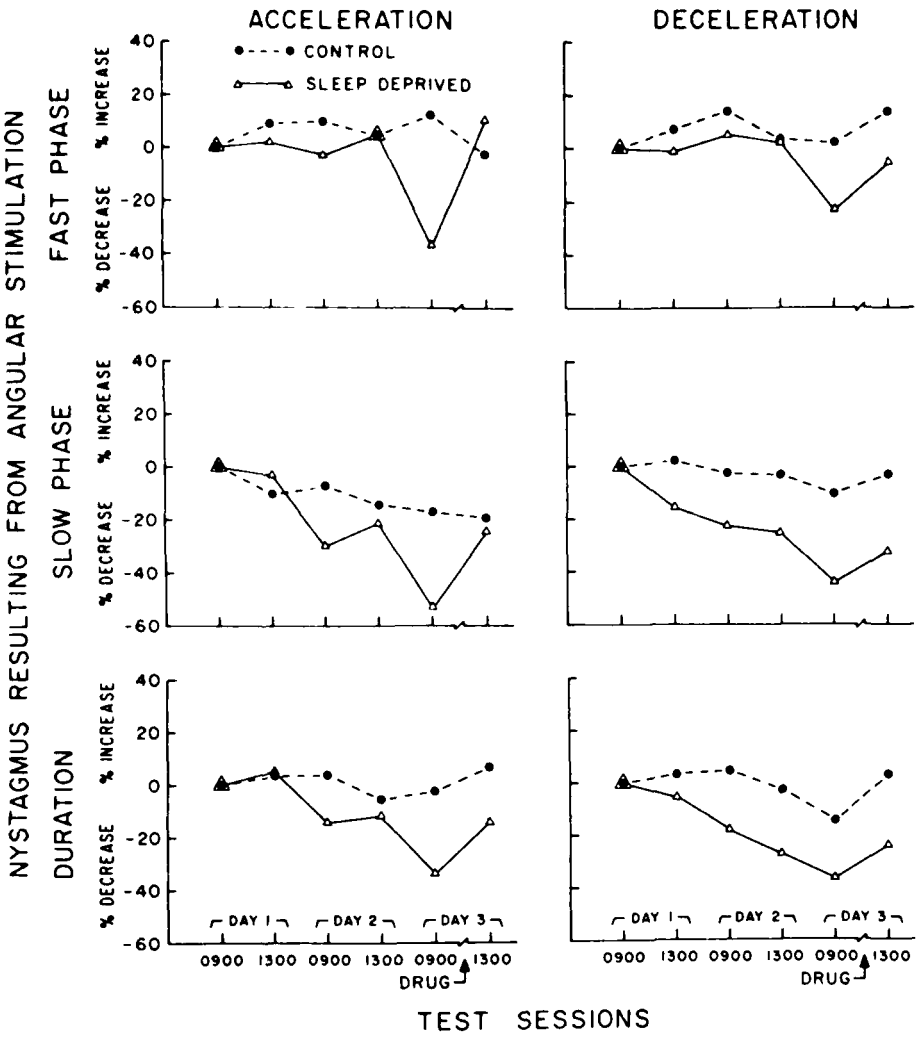

FIGURE 1. CHANGES IN FAST PHASE FREQUENCY, SLOW PHASE DISPLACEMENT, AND DURATION OF VESTIBULAR NYSTAGMUS PRODUCED BY ANGULAR ACCELERATIONS AND DECELERATIONS (5 DEG/SEC^2) ACROSS 6 SESSIONS FOR CONTROL AND SLEEP DEPRIVED SUBJECTS. THE 0 SCORES REPRESENT THE BASE LEVELS (FIRST SESSION) OF OCULAR OUTPUT; SCORES FOR SUCCESSIVE SESSIONS WERE CONVERTED TO PERCENTAGES OF INCREASE OR DECREASE FROM THE BASE LEVELS. THE DRUG (10 MG D-AMPHETAMINE SULPHATE) WAS ADMINISTERED AT 1200 ON DAY 3.

Coriolis Nystagmus. The control group maintained its output of nystagmus for several Coriolis measures (from head movements) or showed only a slight tendency for responses to decline across sessions (see figure 2). The control group showed no significant changes in output of nystagmus for sessions 1 vs 5 to either tilts or return movements of the head (see table 2). Ingestion of d-amphetamine had minimal effects; it resulted in a significant ($p < .05$) drop (compared with session 1) in slow phase output during head tilts, and it

TABLE 1.-RESULTS OF MULTIVARIATE ANALYSES (MANOVAs) FOR MEASURES OF OCULAR NYSTAGMUS AND MEASURES OF PERCEIVED MOTION FROM SESSION 1 (DAY 1, 0900), SESSION 5 (DAY 3, 0900) AND THE D-AMPHETAMINE (DRUG) SESSION (DAY 3, 1300)

		Ocular Nystagmus			Perceived Motion			
Condition	Measures	Groups (G)	Sessions (S)	Interaction (G x S)	Measures	Groups (G)	Sessions (S)	Interaction (G x S)
Accel	Duration	0.11	3.92*	2.90	Duration	2.02	0.54	0.23
	Slow Phase	0.09	7.83**	4.36*	Displacement	2.68	1.90	0.06
	Fast Phase	0.00	2.18	6.80**	Latency	6.10*	3.06	4.02*
Decel	Duration	0.34	7.58**	2.71	Duration	0.21	2.72	0.34
	Slow Phase	2.04	8.71***	3.82*	Displacement	2.24	0.96	0.40
	Fast Phase	4.10	3.11	2.37	Latency	6.19*	0.05	3.29*
Head Tilts	Duration	0.91	6.10**	3.86*	Duration	0.73	2.01	0.52
	Slow Phase	0.94	2.63	4.47*	Displacement	0.53	6.20**	0.10
	Fast Phase	1.24	3.40*	4.39*				
Head Returns	Duration	0.03	0.17	0.07	Duration	0.56	0.88	0.75
	Slow Phae	0.60	1.83	0.90	Displacement	2.10	1.54	1.06
	Fast Phase	2.94	3.18	0.39				

* $p < .05$ ** $p < .01$ *** $p < .001$

TABLE 2.-LEVELS OF STATISTICAL SIGNIFICANCE FOR DIFFERENCES BETWEEN MEASURES OF OCULAR NYSTAGMUS AND MEASURES OF PERCEIVED MOTION FROM SESSION 1 (DAY 1, 0900), SESSION 5 (DAY 3, 0900) AND THE D-AMPHETAMINE (DRUG) SESSION (DAY 3, 1300)

		OCULAR NYSTAGMUS						PERCEIVED MOTION					
		SLEEP-DEPRIVED			CONTROL			SLEEP-DEPRIVED			CONTROL		
Condition	Sessions	Duration	Slow phase	Fast phase	Duration	Slow phase	Fast phase	Duration	Displacement	Latency	Duration	Displacement	Latency
Accel	1 vs 5	.01	.01	.01						.01			
	D vs 5	.05	.01	.01						.01			
	D vs 1												
Decel	1 vs 5	.01	.01	.01						.01	.05		
	D vs 5			.01	.05								
	D vs 1	.01	.01				.05				.05		
Tilt Right (Climb)	1 vs 5	.01	.01	.01					.05	---	.05		---
	D vs 5	.01		.01		.05				---			---
	D vs 1								.01	---	.05		---
Return Left (Dive)	1 vs 5		.05							---			---
	D vs 5						.05			---			---
	D vs 1						.05			---			---

Table 3.-RESULTS OF MULTIVARIATE ANALYSES FOR MEASURES OF OCULAR NYSTAGMUS AND MEASURES OF PERCEIVED MOTION FROM SESSION 1 THROUGH SESSION 5

		Ocular Nystagmus			Perceived Motion			
Condition	Nystagmus Measures	Groups (G)	Sessions (S)	Interaction (G x S)	Subjective Measures	Groups (G)	Sessions (S)	Interaction (G x S)
Accel	Duration	0.69	8.58***	6.00***	Duration	0.50	0.91	1.66
	Slow Phase	0.53	8.29***	1.96	Displacement	1.97	0.50	1.23
	Fast Phase	0.02	2.57*	4.71**	Latency	2.66	3.77**	4.98***
Decel	Duration	0.31	7.10***	1.96	Duration	0.14	3.07*	1.35
	Slow Phase	1.58	4.61**	1.65	Displacement	2.22	2.18	0.38
	Fast Phase	2.88	2.86*	1.39	Latency	4.91*	0.13	2.57*
Head Tilts	Duration	0.03	3.62**	1.75	Duration	1.00	0.71	0.56
	Slow Phase	1.72	1.00	1.37	Displacement	0.28	2.89	0.92
	Fast Phase	3.04	0.80	1.38				
Head Returns	Duration	0.13	0.55	0.74	Duration	0.43	0.91	0.57
	Slow Phase	1.29	1.26	1.20	Displacement	1.55	2.27	0.47
	Fast Phase	2.06	0.65	0.28				

* $p < .05$ ** $p < .01$ *** $p < .001$

TABLE 4.-LEVELS OF STATISTICAL SIGNIFICANCE FOR DIFFERENCES BETWEEN MEASURES OF OCULAR NYSTAGMUS AND MEASURES OF PERCEIVED MOTION FROM ANGULAR ACCELERATIONS DURING SESSION 1 (DAY 1, 0900) THROUGH SESSION 5 (DAY 3, 0900)

		OCULAR NYSTAGMUS						PERCEIVED MOTION					
		SLEEP-DEPRIVED			CONTROL			SLEEP-DEPRIVED			CONTROL		
Condition	Sessions	Duration	Slow phase	Fast phase	Duration	Slow phase	Fast phase	Duration	Displacement	Latency	Duration	Displacement	Latency
Accel	1 vs 2										.05		
	1 vs 3		.05								.05		
	1 vs 4										.05		
	1 vs 5	.01	.01	.01						.01			
	2 vs 3	.05	.05										
	2 vs 4	.05											
	2 vs 5	.01	.01	.01					.05	.01			
	3 vs 4												
	3 vs 5	.01		.01					.05	.01			
	4 vs 5	.01	.01	.01					.05	.01			
Decel	1 vs 2												
	1 vs 3										.05		
	1 vs 4	.01											
	1 vs 5	.01	.01					.05		.05	.01		.05
	2 vs 3												
	2 vs 4	.01											
	2 vs 5	.01	.05					.01					
	3 vs 4												
	3 vs 5			.05					.05	.05			
	4 vs 5			.05					.05	.05		.05	

TABLE 5.-LEVELS OF STATISTICAL SIGNIFICANCE FOR DIFFERENCES BETWEEN MEASURES OF OCULAR NYSTAGMUS AND MEASURES OF PERCEIVED MOTION FROM CORIOLIS STIMULATION DURING SESSION 1 (DAY 1, 0900) THROUGH SESSION 5 (Day 3, 0900)

		OCULAR NYSTAGMUS						PERCEIVED MOTION			
		SLEEP-DEPRIVED			CONTROL			SLEEP-DEPRIVED		CONTROL	
Condition	Sessions	Duration	Slow phase	Fast phase	Duration	Slow phase	Fast phase	Duration	Displacement	Duration	Displacement
Tilt Right (Climb)	1 vs 2	.05									
	1 vs 3		.05						.05		
	1 vs 4		.05						.01		
	1 vs 5	.01	.01	.01					.05	.05	
	2 vs 3										
	2 vs 4								.01		
	2 vs 5										
	3 vs 4										
	3 vs 5										
	4 vs 5										
Return Left (Dive)	1 vs 2					.05					
	1 vs 3										
	1 vs 4								.05		.05
	1 vs 5								.05		
	2 vs 3					.05					
	2 vs 4					.05					
	2 vs 5										
	3 vs 4	.05									
	3 vs 5										
	4 vs 5										

produced an increase in fast phase output during return head movements that made the postdrug score for frequency of nystagmus significantly greater than those of both sessions 1 and 5 ($p < .05$ in both cases).

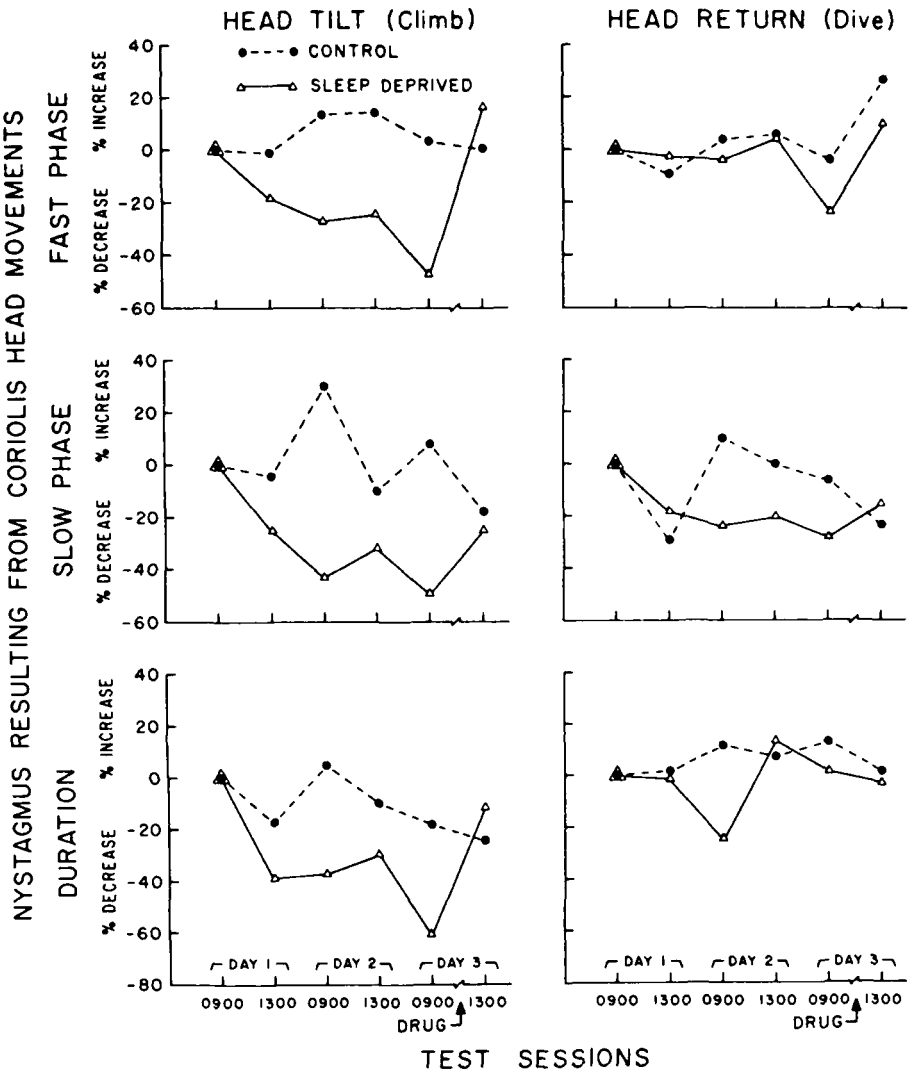

FIGURE 2. CHANGES IN FAST PHASE FREQUENCY, SLOW PHASE DISPLACEMENT, AND DURATION OF CORIOLIS VESTIBULAR NYSTAGMUS PRODUCED BY ACTIVE HEAD MOVEMENTS (A TILT 30 DEG TO THE RIGHT; A TILT 30 DEG TO THE LEFT TO RETURN THE HEAD TO UPRIGHT) DURING CW ROTATION AT 90 DEG/SEC. DESIGNATIONS IN THE GRAPHS ARE THE SAME AS IN FIGURE 1.

For the sleep-deprived group, nystagmus scores from head tilts dropped sharply and regularly across predrug sessions; scores for return-to-upright movements of the head showed less consistent and weaker tendencies to decline. Significant reductions in nystagmic output for sessions 1 vs. 5 occurred for all measures ($p < .01$) during head tilts, and only for the slow phase measure ($p < .05$) during head returns. Although the drug sufficiently elevated ocular

output in all cases of significantly reduced responses so that the postdrug session measures showed no significant differences from session 1, only the duration and fast phase frequency measures for head tilts rose significantly ($p < .01$) above those for session 5.

Turning Experiences. The duration of turning experiences occasioned by angular accelerations and decelerations showed declining trends for both groups of subjects (see figure 3). However, only the deceleration stimulus for the control group yielded a significant ($p < .05$) decline for session 1 vs 5. (Duration scores for sessions 2, 3, and 4 all declined significantly from session 1 for acceleration stimuli for the control group, but the duration score was considerably elevated in session 5. This elevated score might, for this measure, lead to an underestimation of the effect of sleep loss.) The ingestion of d-amphetamine produced no consistent or significant effect, slightly elevating duration scores for the sleep-deprived, and slightly lowering them for the control group.

The amount of turning experienced during accelerations and decelerations showed almost no change across sessions for control subjects. There was a drop (that was short of being statistically different from session 1) only

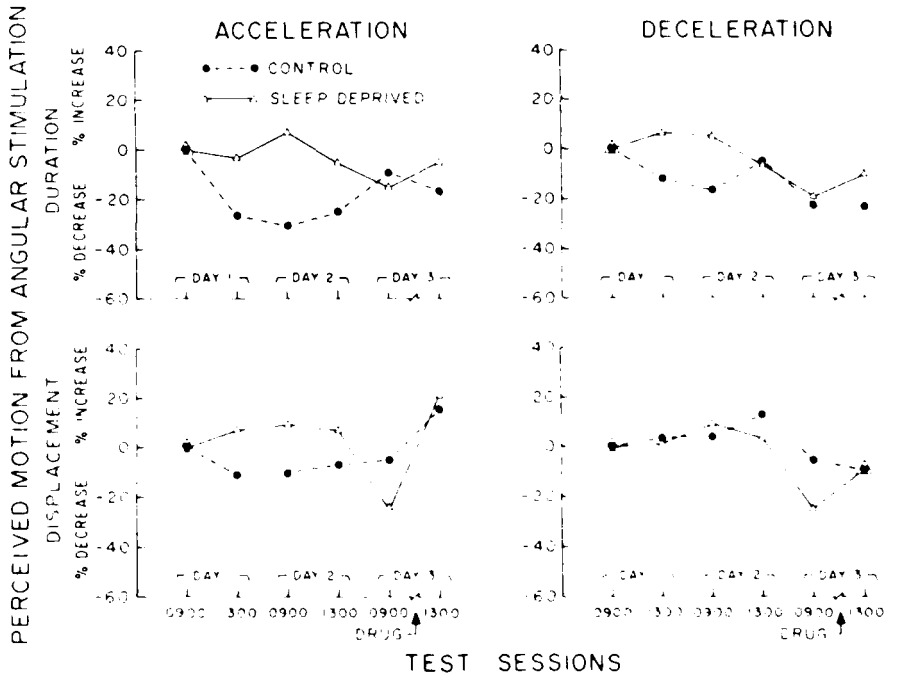

FIGURE 3. CHANGES IN THE DURATION AND AMOUNT OF TURNING EXPERIENCED AS A RESULT OF ANGULAR ACCELERATIONS AND DECELERATIONS (5 DEG/SEC^2) ACROSS 6 SESSIONS FOR CONTROL AND SLEEP DEPRIVED SUBJECTS. DESIGNATIONS IN THE GRAPHS ARE THE SAME AS IN FIGURE 1.

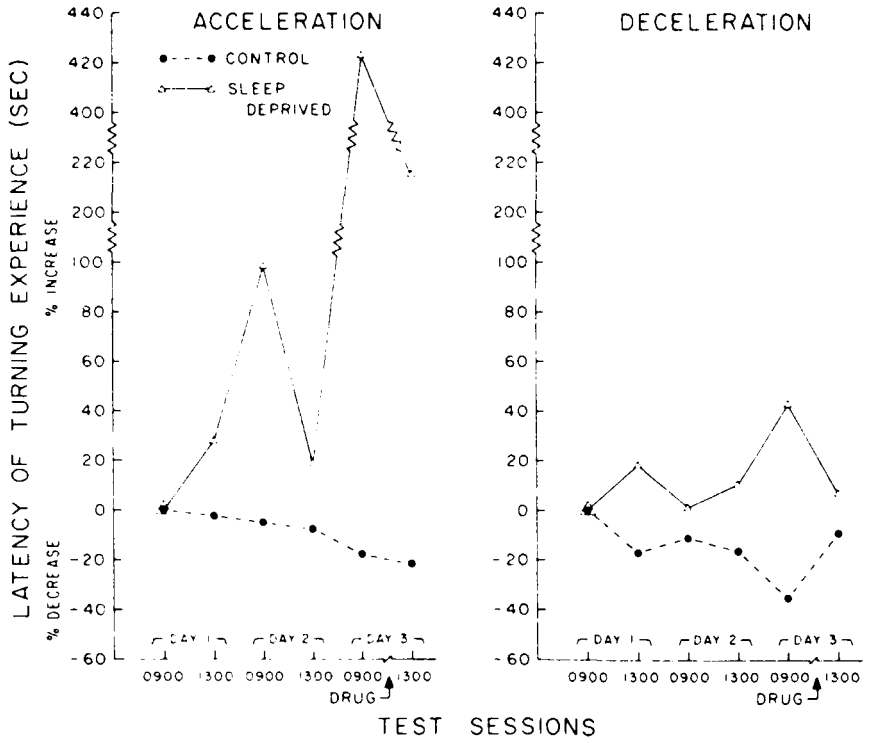

FIGURE 4. CHANGE IN THE LATENCY (THE TIME BETWEEN STIMULUS ONSET AND THE FIRST SIGNAL) OF THE TURNING RESPONSE TO ANGULAR ACCELERATIONS AND DECELERATIONS FOR CONTROL AND SLEEP DEPRIVED SUBJECTS. DESIGNATIONS IN THE GRAPHS ARE THE SAME AS IN FIGURE 1.

during the fifth session for the sleep-deprived group; however, because scores increased somewhat across the first four sessions, the total displacement scores for the fifth session were significantly ($p < .05$) below those of each of the two preceding sessions for the sleep-deprived subjects. The analeptic drug elevated scores above those of session 5 (to both acceleration and deceleration) for the sleep-deprived subjects, but no statistically significant drug-induced changes were obtained. (The postdrug, acceleration-induced, displacement score for the sleep-deprived group increased considerably, but the increase was due largely to an inordinately high signalling rate from one subject. That response represented an increase of approximately 250% over any previous response from that man, whose signalling rate was initially high. His deceleration-induced displacement score was also high, but his nystagmic output was not remarkably affected. Perhaps he was unusually sensitive to the d-amphetamine.)

The latency of signalling turning experiences declined regularly, but not significantly, for control subjects. For sleep-deprived subjects, latency scores tended to increase across sessions and were significantly longer for session 5 vs session 1 ($p < .01$ for both acceleration and deceleration). d-Amphetamine reduced signal latencies for the sleep-deprived and had no reliable effect on the performance of control subjects.

Coriolis Experiences. For both groups, the duration of the Coriolis "climbing" (pitch up) and "diving" (pitch down) experiences showed shallow declining trends that did not reach statistical significance. The ingestion of d-amphetamine produced no noticeable effect on this measure. (See figure 4.)

For both groups, the amount of perceived displacement during head tilts ("climb") showed a relatively regular decline across sessions. The decline from session 1 to 5 was significant for both groups ($p < .05$). Administration of d-amphetamine produced no discernable effect (scores dropped further) and so the postdrug displacement scores were also significantly below those of session 1 ($p < .05$ for the control group; $p < .01$ for the sleep-deprived). For return movements of the head ("diving" sensations), displacement scores tended to increase during the first four sessions and then declined slightly during session 5. Differences between sessions 1 and 5 were not significant (and were numerically higher for session 5) and the drug produced no reliable effect on these displacement measures (control group scores rose; the scores for the sleep-deprived declined).

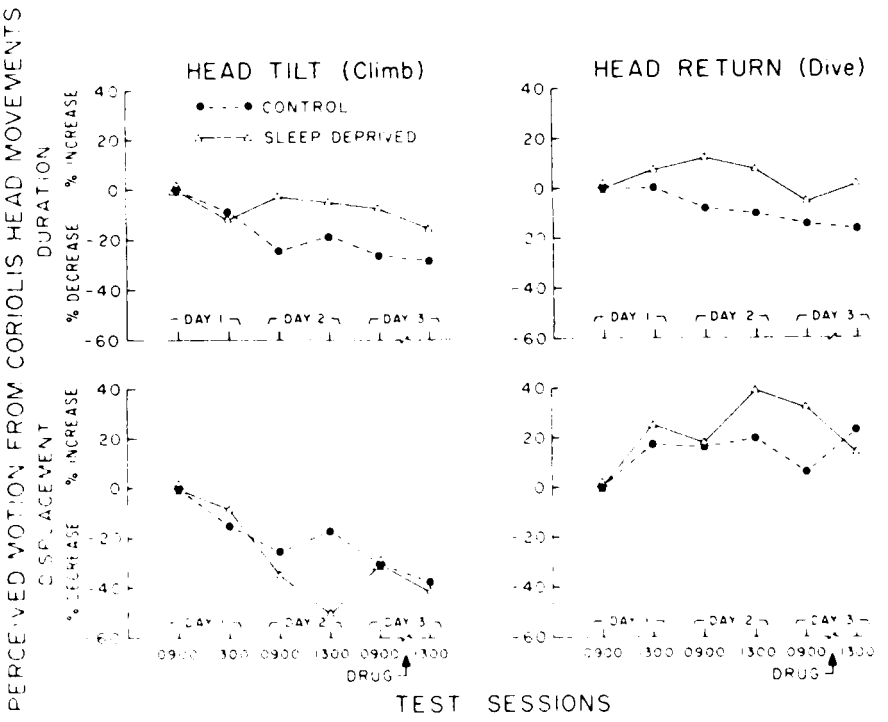

FIGURE 5. CHANGES IN THE DURATION AND AMOUNT OF DISPLACEMENT EXPERIENCED AS A RESULT OF ACTIVE TILT (RIGHTWARD) AND RETURN (LEFTWARD TO UPRIGHT) HEAD MOVEMENTS DURING CW ROTATION AT 90 DEG/SEC. DESIGNATIONS IN THE GRAPHS ARE THE SAME AS IN FIGURE 1.

DISCUSSION

These results show that the ordinary effects of sleep loss on vestibular responses to a moderate angular stimulus are: (i) negligible effects on quantified experiences of turning, (ii) a general decline in slow phase and duration measures of nystagmus, and (iii) no reductive effect on fast phase nystagamus until sometime between 30-50 hrs (i.e., subsequent to the Day 2 session at 1300) of sleep deprivation. These findings agree with the nystagmus results obtained by Wolfe and Brown[12] to their "moderate" stimulus (8 deg/sec^2) and extend from 24 to 50 hrs the period over which the nystagmic response during sleep deprivation is described.

Although Wolfe and Brown[12] had their subjects signal the onset and conclusion of their rotary responses, they reported only latency data and found no pre-to-posttest change (to either their 8 deg/sec^2 or 24 deg/sec^2 stimuli) for a 24-hr period of sleep deprivation. Our results show a small but steady reduction in latency (i.e., quicker responses) for control subjects and increasing latencies for the sleep deprived. Two factors in our latency data for the sleep deprived are worth noting: (i) with latencies during the first two sessions averaging about 5.5 sec, the marked increase during accelerations on the mornings of Days 2 and 3 are largely attributable to two subjects (Day 2) whose latencies exceeded 13 sec and to 3 subjects (Day 3) whose latencies exceeded 15.5 sec. This gradual increase probably reflects the individual variability in response to sleep deprivation since latency scores do not show a rise for all sleep deprived subjects until the afternoon of Day 2. (ii) Latencies for the sleep deprived to accelerations showed markedly more session-to-session variability and proportionately greater increases than did latencies to decelerations. This difference may be attributable to arousal (or to potentiating effects in sleep deprivation) induced by the positive acceleration followed by the Coriolis stimulation shortly before the decelerations were introduced. There was no indication of any consistent differences for accelerations vs. decelerations in responses from the control subjects.

The above rationale for explaining variations in our latency data may also account for the lack of change noted by Wolfe and Brown[12] in their latencies after 24 hrs of sleep deprivation, viz. their stronger stimuli may have been sufficiently more arousing as to prevent a noticeable lengthening of response time for the proportionately few subjects (as suggested by our data) who would have been negatively affected by 24 hrs of sleep deprivation.

Coriolis stimulation caused by rightward head tilts during CW rotation produced a pattern of nystagmus across sessions that was similar to, but more exaggerated than, the results obtained from angular accelerations. That is, all three measures of nystagmus declined over sessions for sleep deprived subjects while the control group showed a declining tendency for slow phase and duration measures and a tendency for increased fast phase output. Moving the head back to an erect position (i.e., a leftward movement during CW rotation) produced nystagmus that showed no clear tendency to decline for control subjects and that seemed mostly resistant to reduction for the sleep deprived; for the latter group (i) only slow phase measures were consistently below the first session's output (and most of that decline occurred from the

morning to the afternoon session on the first day) and (ii) the fast phase output showed no drop until the morning of the third day (i.e., after sometime between 30-50 hrs of sleep deprivation).

These Coriolis findings are not directly comparable to Dowd's[4] results since Dowd (i) used Air Force pilots who were regarded as having been habituated to vestibular stimulation, (ii) reported only one direction of nystagmus (equivalent to our rightward head tilt with its "climbing" sensation), and (iii) used passive movement (the subject's chair was tilted). His findings were different than ours in that 24 hrs of sleep deprivation yielded an increased response (which he accounted for by sleep deprivation having interferred with the vestibular habituation process) in measures that involved slow phase and duration scores.

With regard to motion experiences, angular accelerations produced a slight shortening of the duration of perceived turning across sessions for both the control and sleep deprived groups and had no effect on the amount of turning for either group until the morning of Day 3 when, after 50 hrs of sleep deprivation, the sleep deprived group showed a significant drop.

The "climbing" and "diving" experiences produced by the Coriolis stimuli yielded patterns across sessions that were different from each other, but similar to nystagmic responses to the same stimuli for the two groups of subjects. Thus, the duration (slightly) and the amount (more so) of experienced "climb" declined, while the "diving" displacement increased across sessions and its duration showed little tendency to decline over the 50-hr period. This resistence across sessions to reduction of the "diving" experience (and the accompanying nystagmus) may well relate to the compelling nature of the "diving" sensation; that experience is much more profound than either the "climbing" or turning sensation and is sometimes associated with transient fear (of "falling") on the part of the subject.[1]

The introduction of d-amphetamine after 54 hrs of sleep deprivation produced no clear effects on the various subjective measures of turning and Coriolis displacement for control subjects, but produced increments in all turning scores and shortening of the latency of the subjective response for the sleep deprived. Those effects suggest an influence on arousal that restores some response for the sleep deprived but does not affect reductions (habituation) that may be due to repeated stimulation.

The effect of d-amphetamine on nystagmus from sleep deprived subjects was to increase all measures of the reduced responses to both angular accelerations and Coriolis stimulation. Effects of the drug on control subjects were less consistent. In most cases, responses continued to decline (e.g., for all measures of Coriolis nystagmus except fast phase responses during leftward head movements); in other cases (e.g., for nystagmus during angular accelerations), slight increments were obtained on some measures and decrements on others.

These results provide no evidence for potentiation of vestibular nystagmus as a consequence of sleep loss as proposed by Wolfe and Brown.[12] That interesting proposal, an extrapolated hypothesis from work in various fields by several authors,[3,6,7,9,11] included the notion of a common neural mechanism involved with Stage-1 REM (rapid eye movement) sleep and fast phase nystagmic eye

movements elicited during vestibular stimulation. While there is clear evidence that vestibular nystagmus can occur during REM stages of sleep,[9,10] and that sleep deprivation (and, therefore, "REM deprivation") can increase the amount of REM when sleep finally occurs,[3] the data supporting a potentiating effect of REM deprivation on vestibular nystagmus while subjects are awake (but sleep deprived) were only from one of Wolfe and Brown's[12] two stimuli (24 deg/sec^2) and not the other (8 deg/sec^2). Moreover, there was no control group, and the authors discounted the idea that repetitive stimuli would produce anything other than a decrement in nystagmus measures; with respect to the latter, there are data to the contrary for fast phase responses.[2] Evidence from the present study suggests no sleep loss related potentiation of nystagmus even for periods longer than those used by Wolfe and Brown.[12] However, Wolfe and Brown's stimulus was considerably stronger than that used in the present study and differences in findings might be related to the difference in stimulus levels. In any event, it is clear that considerably more information on vestibular responsivity during sleep deprivation is needed to provide an adequate description of the interactive effects; to establish peripheral evidence of relationships between REM deprivation, sleep deprivation, and fast phase vestibular eye movements; and to define the potential effects of sleep loss in aviation environments, particularly with respect to disorientation.

CONCLUSIONS

Ocular Nystagmus. Sleep deprivation produced a general decline in all measures of nystagmus produced by angular accelerations. In addition, Coriolis (vertical) nystagmus, declined over time during head tilts ("climbing" sensations) but only for sleep deprived subjects. Ocular responses were unaffected during movements which returned the head to an upright position ("diving" sensations).

d-Amphetamine increased those nystagmic responses of the sleep-deprived group that had declined due to sleep loss. The drug produced no reliable changes in responses of the control group.

Motion Experiences. Effects obtained after about 50 hrs of sleep deprivation were generally negligible for the duration and magnitude both of left- and right-turning sensations and of Coriolis ("climbing" and "diving") experiences. However, a pronounced increase occurred for sleep-deprived subjects in the latency of signaling the onset of turning experiences. This increase suggests that performance capabilities, rather than vestibular experiences, were most seriously affected by sleep loss: viz, reaction times were lengthened.

d-Amphetamine produced no reliable changes for the control or sleep deprived groups in subjective vestibular responses (although measures of the turning experiences of the sleep deprived increased somewhat), but the drug shortened the latency (reaction time) for signals of the onset of turning by the sleep deprived.

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