

The Efficacy of Dextroamphetamine for Sustaining Helicopter Pilot Performance: An In-Flight Evaluation

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SUMMARY

The capability to operate 24 hours per day on the battlefield creates a tactical advantage over enemy forces. However, staffing shortages necessitating long work hours devoid of sleep eventually produce overwhelming fatigue, impairing performance and safety. In these situations, the only effective means to sustain performance may be the administration of stimulants. Unfortunately, studies of stimulants such as dextroamphetamine on the actual flight performance of aviators are virtually nonexistent. The present study assessed actual in-flight performance, mood, and alertness of UH-60 pilots during sleep-deprivation periods in which they were given either a 10-mg dose of Dexedrine or a placebo at 0000, 0400, and 0800 within the last 23 hours of each period. Results indicated better control (smaller RMS errors) of several flight parameters (i.e., heading, altitude, airspeed, etc.) under Dexedrine than placebo during straight-and-levels, climbs, descents, right turns, and a left-descending turn. Tendencies toward Dexedrine-related improvements also occurred in the left turns and the Instrument Landing System approach. The Profile of Mood States revealed reductions in fatigue, confusion, and depression concurrent with increases in vigor as a function of Dexedrine. Electroencephalographic data indicated enhanced central nervous system arousal under Dexedrine relative to placebo. No significant side effects occurred. It can be concluded that dextroamphetamine effectively sustained aviator performance during short-term sustained operations.

BACKGROUND

Because sustained operations make it difficult for aviators to receive adequate sleep during combat, the military is exploring countermeasures to offset problems associated with sleep debt. Pharmacological measures may be the only viable alternative in some situations, and the stimulant Dexedrine appears to be very promising for this purpose (1).

Senechal (2) reported that EF-111A Raven jet crews who were administered 5 mg Dexedrine during an Air Force strike on Libya experienced positive effects in terms of overcoming the fatigue of the mission itself and the sleep deprivation which occurred during preparation for the mission. There were no in-flight or landing problems, and all of these electronic-jamming aircraft returned safely to base. Cornum (3) reported that dextroamphetamine also was used with 35 F-15C pilots who were flying combat air patrol missions during Operation Desert Shield/Storm. These pilots were not only flying long missions (6-11 hours), but were sleep deprived and suffering from circadian desynchronization as well. Pilots were issued 5-6

dextroamphetamine tablets (5 mg) at the beginning of flights and were told to self-administer one tablet every 2-4 hours as needed to maintain alertness. The aviators reported clear benefit from the drug, and the unit commander concluded that dextroamphetamine administration contributed significantly to the safety of operations. There were no reported adverse effects, even in personnel who took 10 mg at a time, and no aviators reported a need to continue the drug once proper work/sleep schedules were reinstated.

Emonson and Vanderbeek (4) indicated that Air Force pilots effectively used dextroamphetamine during Operation Desert Storm to maintain acceptable performance during continuous and sustained missions. The medication was found to be both safe and beneficial in terms of overcoming fatigue without producing unwanted side effects. These results were later supported by Cornum, Cornum, and Storm (5) who surveyed F15-C squadrons deployed in Operation Desert Shield. Fifty-seven percent of respondents indicated they used dextroamphetamine, mostly on long, low-task, night missions. This medication was considered beneficial in terms of flight safety without inducing the feelings of hyperactivity associated with caffeine.

These anecdotal reports have been supported in controlled laboratory investigations of the effects of Dexedrine on the simulator flight performance of sleep deprived aviators (6,7). These investigations were placebo-controlled studies of 12 Army helicopter pilots who completed UH-60 simulator flights, psychological evaluations, and electrophysiological assessments throughout 36-hour periods of continuous wakefulness. Flights occurred at 0100, 0500, 0900, 1300, and 1700. One hour prior to each of the first three flights, aviators were given 10 mg of Dexedrine or placebo. Dexedrine improved aviator control on the majority of flight maneuvers including descents, straight-and-levels, standard-rate turns, low-level navigation, and a left-descending turn. Performance was not enhanced on hovering turns or formation flight. Dexedrine most noticeably facilitated flight performance at 0500, 0900, and 1700 (after 22, 26, and 34 hours of continuous wakefulness). Slow-wave electroencephalographic (EEG) activity and ratings of fatigue and confusion were reduced after Dexedrine administration, indicating a positive effect on general alertness. Although recovery sleep after Dexedrine was somewhat compromised, there were no clinically significant behavioral or physiological effects. Thus, it appeared that Dexedrine was safe and effective for sustaining helicopter pilot performance during short periods of sleep loss. However, a definitive conclusion about sustaining flight performance with Dexedrine required actual in-flight investigation.

METHODS

Subjects

Ten UH-60 pilots (between the ages of 28 and 36, with a mean age of 31.9) were tested. Five used tobacco (only during breaks between sessions), but none used alcohol or other drugs during the protocol.

Apparatus

Drug administration. At dose times on Dexedrine days, subjects received 2 capsules, each containing 5 mg Dexedrine. On placebo days, subjects received matching capsules containing lactose.

UH-60 helicopter. Flights were conducted in a specially-instrumented Sikorsky UH-60A helicopter. Aspects of aircraft control including heading, airspeed, slip, roll, vertical speed, and altitude control were recorded by computer during flights.

EEG evaluations. EEG activity (from Fz, C3, Cz, C4, Pz, O1, and O2) was collected between flights with a Cadwell Spectrum 32. The low filter was set at 0.53 Hz and the high filter was set at 70 Hz. Electrodes were secured to the subjects' scalps with collodion.

Profile of Mood States (POMS). Subjective ratings of tension-anxiety, depression-dejection, anger-hostility, vigor-activity, fatigue-inertia, and confusion-bewilderment were made with the POMS.

Procedure

Tests were conducted at 0900, 1300, and 1700 on Monday, Tuesday, and Thursday; and at 0100, 0500, 0900, 1300, and 1700 on Wednesday and Friday (the sleep deprivation periods). On Wednesday and Friday, drug or placebo doses were administered to subjects at 0000, 0400, and 0800. At dose times, subjects received either 10 mg Dexedrine or placebo. Sessions began with a flight in the UH-60 helicopter and ended with a cognitive test (not reported here).

UH-60 flights. Each flight was 30 minutes in length and consisted of straight and levels, standard-rate turns, climbs and descents, and an ILS. During each maneuver, subjects maintained control parameters (headings, altitudes, airspeeds, etc.), based upon safety-pilot instructions. The first several maneuvers were conducted with the UH-60's automatic flight-path control system (AFCS) engaged, and the remaining maneuvers were flown without the AFCS. Root mean square (RMS) errors were calculated for each control parameter during each maneuver.

EEG evaluations. Evaluations were conducted after flights. Data were recorded for 1.5 minutes with eyes open and 1.5 minutes with eyes closed. Power was calculated for delta (1.5-3.0 Hz), theta (3.5-8.0 Hz), alpha (8.0-13.0 Hz), and beta (13.0-20.0 Hz) bands based upon 3, artifact-free 2.5 second epochs.

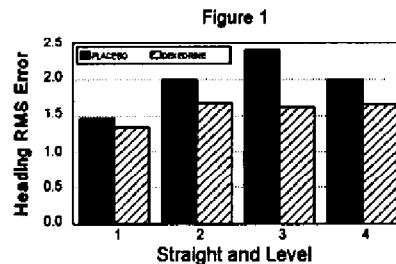
POMS. The POMS was administered about 45 minutes after the EEG. Subjects completed a checklist in which they indicated how well each of 65 mood adjectives described their present feelings. The data were scored to produce ratings on six factors.

RESULTS

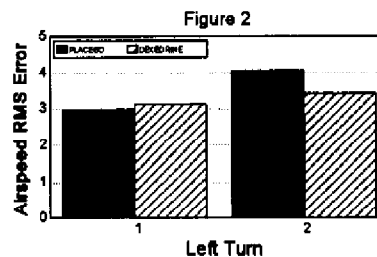
Flight performance

The effects of drug (Dexedrine, placebo) and time (0100, 0500, 0900, 1300, and 1700) on RMS errors were analyzed with analysis of variance (ANOVA). Only drug-related effects are reported here.

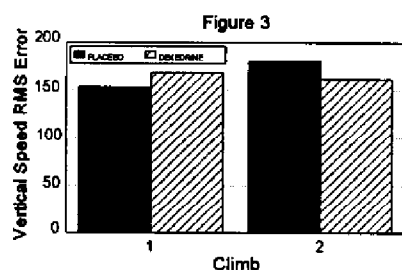
Straight and levels. Analysis of heading, altitude, airspeed, slip, and roll control in the four iterations of straight-and-level (SL) flight, indicated a drug-by-iteration interaction on heading ($F(1,9)=18.36$, $p<.01$) and drug main effects on heading ($F(1,9)=19.79$, $p<.01$) and airspeed ($F(1,9)=5.24$, $p=.05$). The interaction was due to larger errors under placebo than Dexedrine during SLs 2-4, with no difference in SL 1 (see figure 1). Drug main effects were due to decreased errors under Dexedrine versus placebo. Heading errors were 1.6 and 2.0 degrees respectively, and airspeed errors were 2.9 and 3.2 knots.



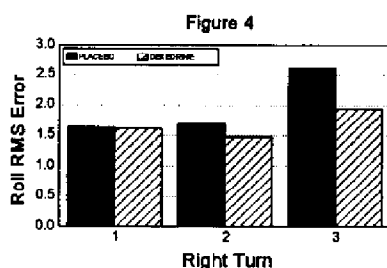
Left standard-rate turns. Analysis of turn rate, altitude, airspeed, slip, and roll control in the two left turns showed two marginal effects--a drug-by-iteration interaction on airspeed ($F(1,9)=4.37$, $p=.07$) and a drug main effect on roll ($F(1,9)=3.26$, $p=.10$). As shown in figure 2, the interaction was due to larger airspeed errors under placebo than Dexedrine in the second turn (conducted without the AFCS). The main effect on roll was due to larger errors under placebo than Dexedrine (2.1 versus 1.7 degrees).



Climbs. Analysis of heading, airspeed, slip, roll, and vertical speed in the two climbs revealed a drug-by-iteration effect on vertical speed ($F(1,9)=5.35, p=.05$) and drug main effects on heading ($F(1,9)=6.36, p=.02$) and slip errors ($F(1,9)=6.02, p=.04$). The interaction was due to an unexpected increase in errors under Dexedrine in the first but not the second climb (see figure 3). The drug main effects were due to smaller errors under Dexedrine relative to placebo. Heading errors were 1.5 and 1.7 degrees, and slip errors were .21 versus .25 ball widths.



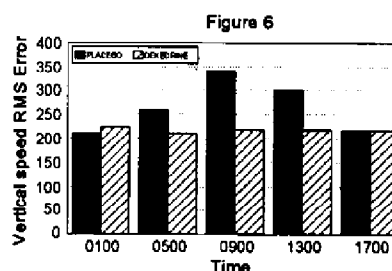
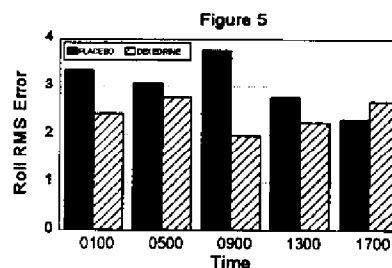
Right standard-rate turns. The ANOVA on turn rate, altitude, airspeed, slip, and roll control in the three right turns indicated a drug-by-iteration interaction on roll control ($F(2,18)=3.54, p=.05$). This was due to larger RMS errors under placebo than Dexedrine during only the third turn (conducted without the AFCS). This interaction is depicted in figure 4.



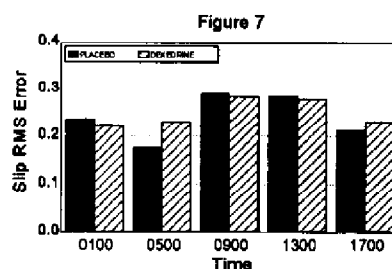
Descents. Analysis of heading, airspeed, slip, roll, and vertical speed control in the two descents indicated drug main effects on heading ($F(1,9)=5.64, p=.04$), airspeed ($F(1,9)=5.44, p=.04$), roll ($F(1,9)=9.98, p=.01$), and vertical speed control ($F(1,9)=9.90, p<.01$). All were due to smaller RMS errors under Dexedrine than placebo--means were 1.6 versus 1.8 degrees of heading, 3.0 versus 3.4 knots of airspeed, 1.3 versus 1.6 degrees of roll, and 192 versus 224 feet per minute of vertical speed.

Left descending turn. Analysis of turn rate, airspeed, slip, roll, and vertical speed control during the left-descending turn indicated drug-by-time interactions on roll ($F(4,36)=2.87, p=.04$) and vertical speed ($F(4,36)=3.98, p<.01$). In addition, there was a drug main effect on vertical speed ($F(1,9)=7.12, p=.03$). The interactions were due to smaller roll errors under Dexedrine versus placebo at 0900 and a similar effect on vertical speed errors at 0500 and 0900 (see figures 5 and 6). The drug main effect on vertical speed also was due to smaller

errors under Dexedrine versus placebo (225 and 264 feet per minute, respectively).



ILS approach. The ANOVA on airspeed, slip, and roll control, and localizer and glide-slope tracking accuracy on the ILS revealed a drug-by-session interaction on slip control ($F(4,36)=2.94, p=.03$) and glide-slope tracking ($F(4,36)=2.19, p=.09$). Also, there was a drug main effect on localizer tracking ($F(1,9)=3.98, p=.08$). The slip interaction was due to an unexpected increase in errors under Dexedrine at 0500 but not elsewhere (see figure 7). The glide slope interaction was due to smaller errors under Dexedrine at 1300. The drug main effect on localizer tracking was due to better accuracy under Dexedrine than placebo (1.1 and 1.4 dots, respectively).

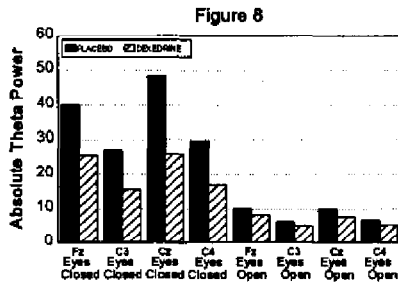


EEG activity

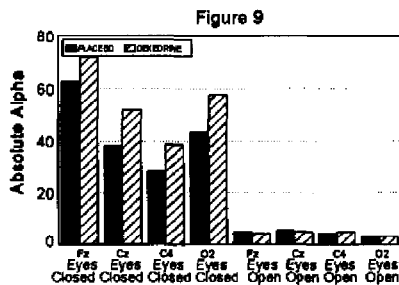
EEG power was assessed with ANOVAs for drug (Dexedrine, placebo), time (0220, 0620, 1020, 1420, and 1820), and eyes (closed, open). The data from 7 electrodes were analyzed separately.

Delta activity. There were no interactions or other drug effects. Although tendencies ($p=.10$) were seen suggesting slight increases in delta at Fz, Cz, C4, and C3, none were statistically significant.

Theta activity. Drug-by-eyes interactions occurred at Fz ($F(1,9)=9.14$, $p=.01$), C3 ($F(1,9)=6.41$, $p=.03$), Cz ($F(1,9)=8.74$, $p=.02$), C4 ($F(1,9)=7.56$, $p=.02$), and marginally at Pz ($F(1,9)=4.80$, $p=.06$) because of greater theta under placebo versus Dexedrine at eyes closed (see figure 8). Drug main effects were found at C3 ($F(1,9)=7.84$, $p=.02$), Cz ($F(1,9)=8.45$, $p=.02$), C4 ($F(1,9)=6.72$, $p=.03$), and Pz ($F(1,9)=6.04$, $p=.04$) because of increased theta under placebo.



Alpha activity. Drug-by-eyes interactions occurred at C4 ($F(1,9)=7.93$, $p=.02$), Cz ($F(1,9)=8.40$, $p=.02$), Fz ($F(1,9)=11.80$, $p<.01$), and O2 ($F(1,9)=6.38$, $p=.03$) due to more alpha under Dexedrine than placebo with eyes closed (see figure 9). Drug main effects at Fz ($F(1,9)=9.93$, $p=.01$), Cz ($F(1,9)=6.51$, $p=.03$), C4 ($F(1,9)=7.01$, $p=.03$), and O2 ($F(1,9)=6.36$, $p=.03$) were also due to increased alpha under Dexedrine.



Beta activity. No drug-related interactions or main effects were observed. Thus, beta activity (13-20 Hz) was unaffected by Dexedrine administration.

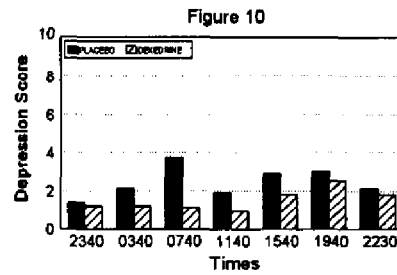
POMS

POMS data were analyzed with ANOVA in which the factors were drug (Dexedrine, placebo) and session (2340, 0325, 0725, 1125, 1525, 1925, and 2225). Each of the six factors was examined separately.

Tension-anxiety scale. There were no interactions or main effects which would reflect differences in musculoskeletal tension under placebo versus Dexedrine.

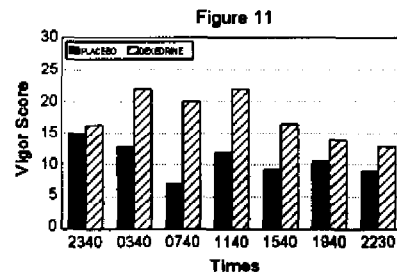
Depression-dejection scale. Despondence and sadness was affected by the combination of drug and session ($F(6,54)=2.92$, $p=.02$) in that more depression was seen under placebo relative

to Dexedrine at 0725. There were no drug-related differences at the other times (see figure 10).

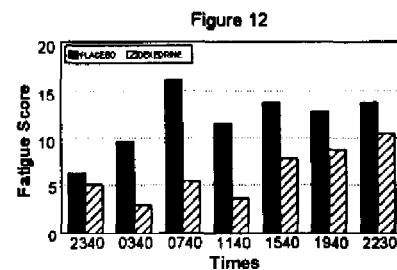


Anger-hostility scale. There were no significant main effects or interactions which would reflect differences in anger and antipathy towards others under placebo versus Dexedrine.

Vigor-activity scale. Energy levels were affected by the combination of drug and session ($F(6,54)=5.05$, $p<.01$) in that vigor scores were lower under placebo than Dexedrine at 0325, 0725, 1125, and 1925, but not at 2340 (before deprivation) or at 2225 (at the end of deprivation). This can be seen in figure 11. A drug main effect ($F(1,9)=46.50$, $p<.01$) was due to reduced vigor ratings under placebo versus Dexedrine (10.7 for placebo and 17.6 for Dexedrine).

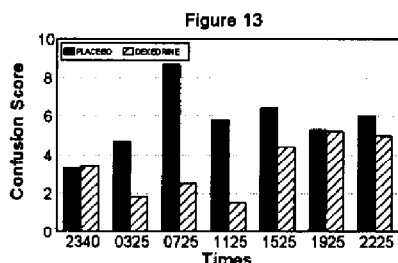


Fatigue-inertia scale. Weariness and tiredness ratings showed a significant interaction between drug and session ($F(6,54)=2.75$, $p=.02$) and a significant effect on the drug factor ($F(1,9)=28.20$, $p<.01$). The interaction was due to higher levels of fatigue under placebo than Dexedrine at 0325, 0725, 1125, and 2225 but not elsewhere (see figure 12). A drug main effect resulted from higher fatigue scores under placebo than Dexedrine (11.9 versus 6.3).



Confusion-bewilderment scale. Scores reflecting increased difficulties in mental abilities showed a drug-by-session interaction ($F(6,54)=4.83$, $p<.01$), and a drug main effect

($F(1,9)=33.51$, $p<.01$). The interaction was due to more confusion under placebo than Dexedrine at 0325, 0725, and 1125, but not at other times (see figure 13). The drug main effect was attributable to a reduction in self-perceptions of confusion under Dexedrine in comparison to placebo (the means were 3.4 versus 5.7).



DISCUSSION

Dexedrine improved flight performance during the final 23 hours of a 40-hour period of continuous wakefulness. Several helicopter control parameters were more precisely maintained under Dexedrine than placebo in the straight and levels, left turns, climbs, descent, left descending turn, and ILS. A similar effect also occurred in one of the right turns. During the left descending turn, Dexedrine was particularly helpful at 0500 and 0900. There were 2 instances out of 19 in which a reversal of the expected drug effects occurred (vertical speed control was better under placebo on the first climb, and slip control was better under placebo at 0500 on the ILS); but these were exceptions. Overall, these results are consistent with those from an earlier simulator study (6,7). However, there were fewer significant in-flight drug effects than simulator effects, probably because of the variance-producing impact of turbulent weather, traffic delays, radio distractions, and environmental temperature changes present in the real aircraft. Also, in-flight-study participants benefitted from the alerting effects of frequent sunlight exposures, periodic walks outside of the Laboratory, and changes in scenery associated with traveling from the Laboratory to the airfield every 4 hours.

EEG changes were consistent with performance effects in that theta activity, which increases as a function of sleep deprivation (8), was reduced by Dexedrine. Since cognitive impairments are related to increased theta (9), changes in this slow-wave EEG suggests Dexedrine-related improvements in performance were partially due to enhancements in central nervous system (CNS) arousal. This interpretation was supported by the fact that EEG alpha activity was increased by the drug in comparison to placebo. Since alpha suppression is an indicator of sleep onset, the alpha increases show that Dexedrine reduced the potential for sleepiness-related performance errors.

Subjective mood states were improved after Dexedrine, especially in the morning and the middle of the day. In comparison to placebo, depression-dejection ratings on the POMS were lower at 0725; fatigue-inertia scores were reduced at 0325, 0725, 1125, and 1925; and confusion-

bewilderment scores were lower under Dexedrine at 0325, 0725, 1125, and 2225. Vigor-activity ratings were improved by Dexedrine from 0325 to 1125 and at 1925. Thus, in addition to improvements in objective performance and alertness under Dexedrine, it is clear that feelings of alertness were sustained by the drug throughout the majority of the sleep-deprivation test sessions. These results are consistent with those of Newhouse et al. (10) and Caldwell, Caldwell, and Crowley (11).

In conclusion, this in-flight evaluation supports the results from simulator studies in which Dexedrine maintained flight performance, CNS arousal, and mood during prolonged wakefulness. These results support previous suggestions that dextroamphetamine should be considered a viable countermeasure for fatigue and sleep deprivation in operational environments (3,5,6,7,11,12). Although Dexedrine produced general cardiovascular stimulation and slight impairments in sleep quality (13), these negative effects are inconsequential compared to the improvements in flight performance, mood, and alertness associated with this medication. Dexedrine, administered prophylactically, is particularly beneficial for preventing dangerous reductions in aviator performance and alertness that are most evident between 0300 and 1000 in the morning. Thus, when sleep deprivation is unavoidable, short-term Dexedrine administration is recommended.

Future research should address the issue of whether longer-term use of dextroamphetamine is a viable option for personnel who may be sleep deprived for 3-4 days. It may be that the short-term benefits disappear after 1-2 days because of sleep-pressure, drug tolerance, or physiological stresses. However, until these factors can be investigated, it may be concluded that Dexedrine is a good countermeasure for sleep deprivation in operations that require up to 40 hours of continuous wakefulness.

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