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Monologue Speech: Effects of *d*-Amphetamine, Secobarbital and Diazepam

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HIGGINS, S. T. AND M. L. STITZER. *Monologue speech: Effects of* d-*amphetamine, secobarbital and diazepam*. PHARMACOL BIOCHEM BEHAV **34**(3) 609–618, 1989.—Drugs of abuse from different pharmacological classes increase social conversation. Alcohol and *d*-amphetamine also increase rates of talking in subjects producing speech monologues in an isolated context. This latter finding suggests that the increases observed during dyadic social conversation may represent general increases in talking and not specific effects on social interaction. The present study was conducted to assess whether other abused drugs also increase monologue speaking. The acute effects of secobarbital (0, 50, 150, 250 mg), *d*-amphetamine (0, 25 mg) (Experiment 1), and diazepam (0, 10, 20, 40 mg) (Experiment 2) were investigated in healthy, adult volunteers. Secobarbital and *d*-amphetamine both increased the total amount of speech emitted, while diazepam generally had no effect or decreased talking. Experiment 3 was conducted to further compare the effects of secobarbital (0, 50, 150, 250 mg) and diazepam (0, 5, 15, 25 mg) using a within-subject, crossover design. Secobarbital increased talking in three of the four subjects studied, while diazepam, again, had no effect or decreased talking. In contrast to the differences noted with talking, secobarbital and diazepam both decreased response rates in a nonverbal performance task (i.e., circular-lights procedure); they also produced many similar effects on various subject-rated measures of drug effect. Thus, the differences in the effects of these two compounds on talking are not the result of a general difference in their overall profile of behavioral effects. In summary, the results obtained with secobarbital and *d*-amphetamine further demonstrate that an explicitly social context is not a necessary condition to observe drug-produced increases in speech quantity. The failure of diazepam to reliably increase talking in the present study illustrates the existence of some pharmacological specificity in the effect of drugs on human speech, and suggests another way in which the behavioral effects of the barbiturates and benzodiazepines may differ.

Secobarbital | *d*-Amphetamine | Diazepam | Human speech | Behavioral effects | Behavioral pharmacology | Drug abuse | Social behavior

DRUGS of abuse from diverse pharmacological classes increase social interaction (4,19). For example, alcohol (45–67 g), *d*-amphetamine (5–30 mg), secobarbital (60–300 mg), and hydromorphone (10–18 mg) increase dyadic social conversation (6, 21, 22). Additionally, when alcoholic subjects are given opportunities to choose between socializing or earning money, they choose the social option significantly more when self-administering alcoholic beverages (89 g/day) as compared to nonalcoholic beverages (7). *d*-Amphetamine (12.5–25 mg) produces a similar increase in preference for social interaction over monetary reinforcement in normal volunteers (14).

Interestingly, alcohol (45–67 g) and *d*-amphetamine (5–20 mg) also increase talking in the absence of a social partner (15,23). In these studies, normal volunteers were instructed to produce naturalistic speech monologues during forty-minute experimental sessions. *d*-Amphetamine and alcohol increased overall rates of talking in a dose-dependent manner. Interestingly, while alcohol increased rates of talking, it decreased response rates on a nonverbal behavioral task (i.e., circular-lights procedure). Thus, the increases in talking were not the result of alcohol producing a nonspecific increase in overall activity levels.

The present study was conducted to further investigate drug effects on monologue talking. Specifically, we investigated the effects of two sedative compounds, secobarbital and diazepam, to

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assess the generality of effects on monologue speech across a wider range of drugs. Our hypothesis was that secobarbital would increase talking since it increases rates of dyadic social conversation, and because its behavioral effects are often similar to those of alcohol, which increases monologue talking (15). Diazepam was included as a sedative with a behavioral profile that sometimes differs from that of the barbiturates and alcohol (11). The effects of diazepam could not be predicted, however, since its effects on social conversation, to our knowledge, have not been previously studied (20). *d*-Amphetamine was included as a positive-control condition since this compound has been previously demonstrated to increase rates of monologue speech. Finally, the circular-lights procedure and various self-report measures were included in this study to provide additional information concerning similarities and differences in the behavioral effects produced by these compounds.

EXPERIMENTS 1 AND 2: BETWEEN-GROUPS COMPARISON

METHOD

Subjects

A total of 13 healthy volunteers participated in these two experiments. Six subjects (3 M and 3 F) participated in Experiment 1 and nine subjects (5 M and 4 F) participated in Experiment 2; two subjects (1 M and 1 F) participated in both experiments. Average age and body weights of subjects in Experiment 1 were 26.4 years (S.D. = 5.9) and 163.2 lbs (S.D. = 29.4), respectively. Average age and body weights of subjects in Experiment 2 were 30.2 years (S.D. = 7.8) and 152 lbs (S.D. = 26.2), respectively. All subjects were medically screened and provided informed consent prior to participation. Subjects were compensated at a rate of approximately $5 per hr of study participation. Subjects were requested to abstain from illicit-drug use during the experiment and no alcohol was to be consumed within 12 hr and no caffeinated beverages or food within two hr of experimental sessions.

Setting and Apparatus

The setting and apparatus were the same in both experiments and have been described previously (15). Subjects were seated facing a console located in a $2.4 \times 2.4 \times 1.2$ m sound attenuated chamber. Vocalizations were detected by sensitive nondirectional microphones (Sony model ECM-16 electret), which clipped onto subjects' clothing. Microphones were interfaced with a voice-operated relay (VOR) and a PDP-8 computer. Speech episodes were defined as 1-sec closures of the VOR and were cumulated to represent total seconds of speech. Isolated switch closures of less than 1 sec were not recorded to minimize inclusion of extraneous sounds (e.g., coughs) in the measure of total seconds of speech. A white session light was located on the console that was illuminated for the duration of the talking sessions and also a blue feedback light that was illuminated upon closure of the VOR. A circular-lights device, which has been described previously (10), was also used in the study and was located in a separate room. Briefly, this device is a wall-mounted panel consisting of 16 button-lights arranged in a circle with a 56 cm diameter. A session start button, session light, and a point counter are located in the center of the panel.

Drugs

Secobarbital sodium (0, 50, 150, 250 mg) and *d*-amphetamine sulfate (0, 25 mg) were studied in Experiment 1. These drug doses were chosen to be comparable to the doses used in our prior studies (13, 14, 23). The single dose of *d*-amphetamine was included as a positive control condition, since that dose increased talking in our prior studies (6,14). In our prior studies with secobarbital and *d*-amphetamine (14,21), we began testing 60 and 120 minutes after drug administration, respectively. To follow the same procedures in the present study, subjects always ingested four size 0 opaque capsules; two capsules were administered at 120 min and two more capsules at 60 min prior to the start of the talking session. On days when secobarbital was tested, the capsules ingested at 120 min always contained only lactose and those ingested at 60 min contained one of the doses of secobarbital noted above. On days when *d*-amphetamine was tested, the capsules ingested at 60 min always contained only lactose and those ingested at 120 min contained either lactose or 25 mg *d*-amphetamine sulfate. This dosing schedule was conducted under double-blind conditions. A minimum of 48 hr elapsed between drug sessions. Subjects were exposed to the single dose of *d*-amphetamine, placebo and each of the secobarbital doses in a mixed order, which was then replicated two more times in each subject; i.e., 3 exposures per dose condition.

Diazepam (0, 10, 20, 40 mg) was studied in Experiment 2. Drug was administered 40 min prior to the start of the talking session in two size 0 opaque capsules under double-blind conditions. A minimum of 72 hr elapsed between drug sessions. Dose-effect curves were determined three times in each subject. Order of exposure to the different doses was mixed.

Procedure

All other experimental procedures were the same in Experiments 1 and 2.

Instructions. Subjects were informed that the effects of sedatives and stimulants on mood and behavior were being studied and that as part of the study they would have to spend some portion of each session providing speech monologues. Prior to the first talking session, subjects received a general set of instructions while seated in the experimental space. They were told that the onset of a white light on the console indicated their talking session had begun and its offset indicated the session was completed. They were instructed that during these sessions their job was to talk, that they could talk about any topic, and that they could talk as much or as little as they liked, but that they did have to speak at least occasionally so we knew they were not sleeping. Humming, singing, whistling, etc., were not permitted and subjects were told that only naturalistic talking was acceptable. Subjects were informed that a blue light on the console would illuminate when the microphone was picking up their voice and that when they were not talking they should remain quiet so that extraneous sounds were not recorded. An attempt was made to reassure subjects that while this exercise may seem peculiar initially, most people adapted quickly and generally talked about things going on in their lives, their daily plans, etc. If subjects failed to speak during the initial session, they were reminded that they could speak as much or as little as they liked, but they did need to speak occasionally so we knew they were not sleeping. If subjects emitted unacceptable vocalizations (e.g., humming) they were reinstructed that only naturalistic speech was acceptable.

Speech measurement. Baseline sessions were conducted daily (Monday–Friday) without drug until seconds of speech per session stabilized, i.e., no discernible increasing or decreasing trends in the data. Stability was usually achieved within 4–5 sessions after which drug testing began. Talking sessions were always 40 min in duration, and to assess within-session changes in talking, seconds of speech during the 0–10-, 11–20-, 21–30-, and 31–40-min

periods of the session were recorded separately.

Circular-lights measure. A 60-sec trial on the circular-lights device was completed predrug and immediately before and after talking sessions. During the 60-sec trial, subjects cumulated points by pressing the circular series of 16 buttons as rapidly as possible in response to the random-sequenced illumination of their associated lights; functionally, this was a fixed-ratio 1 schedule of point presentation across multiple operanda. Subjects started a trial by pressing the start button, which resulted in the illumination of the session light and one of the peripheral lights. Pressing the button associated with the illuminated peripheral light produced a brief feedback tone, incremented the counter by one, and resulted in the illumination of a new peripheral light at a random position. Subjects were instructed to earn as many points as possible using their preferred hand.

Self-reports. The following self-report instruments were completed postsession: 1) a 49-item (T-F) questionnaire, derived from the Addiction Research Center Inventory (ARCI) (18), which consists of five empirically derived subscales purported to measure sedative effects (PCAG), euphoric effects (MBG), dysphoric and psychotomimetic effects (LSD), and amphetamine-like effects (A, BG); 2) the Profile of Mood States (POMS), which is a 65-item five-point adjective rating scale that has been factor analyzed into the following eight clusters: anxiety, depression, anger, vigor, fatigue, confusion, friendliness, and total mood score; 3) a 100-mm visual-analog line on which subjects rated their degree of drug-produced "high" from 0 (not at all) to 100 (highest I've ever been); 4) a set of 5 four-point items on which subjects rated their degree of drug effect, sleepiness, drunkenness, relaxation, and alertness from 1 (mild) to 4 (extreme) and a 5-point scale on which subjects rated their degree of drug liking from 0 (not at all) to 4 (very much). On all of the self-report measures, subjects were instructed to answer based on how they felt during the experimental session.

Data Analysis

Daily data for each subject were averaged across their repeated observations under each dose condition. Individual subject means were used in a two-way repeated measures ANOVA for analyzing effects on talking; drug dose served as one factor and within-session time during the 40-min talking session (i.e., 0–10, 11–20, 21–30, and 31–40) was the second factor. The same analysis was used for the circular-lights measure with dose as one factor and minutes after drug administration as the second factor. A one-way (dose condition) repeated measures ANOVA was used for the ARCI, POMS, and visual-analog "high" scale. Duncan's Multiple Range Test was used in post hoc comparisons of significant effects observed in the ANOVA's. The 4- and 5-point scales on which subjects rated various drug effects (e.g., drug effect, sleepy, etc.) provided ordinal data and thus Friedman's Nonparametric Tests were conducted with those measures, and the method of Hettnansperger was used in post hoc comparisons (12). Across all of the measures, separate analyses were conducted assessing the effects of 1) *d*-amphetamine and placebo, 2) the varying doses of secobarbital and 3) the varying doses of diazepam. Effects were considered significant at $p \leq 0.05$. All statistical analyses were based on absolute scores, although data are shown in figures as percent of placebo values to minimize between-subject differences in absolute scores.

RESULTS

Total Speech

Secobarbital. Total seconds of speech increased as an orderly function of secobarbital dose ($p<0.01$). This effect is shown in the upper panel of Fig. 1 with effects of the active doses represented as percent of placebo values. The 250 mg dose increased total talking significantly above levels observed with the 50 mg and placebo doses. The 150 mg dose increased talking significantly above placebo levels. There were no other significant differences. Inspection of results obtained with individual subjects showed that total speech increased across all six subjects after both the 150 mg and 250 mg doses; however, the increases in speech were of larger magnitude after the 150 mg dose than the 250 mg dose in three of the six subjects.

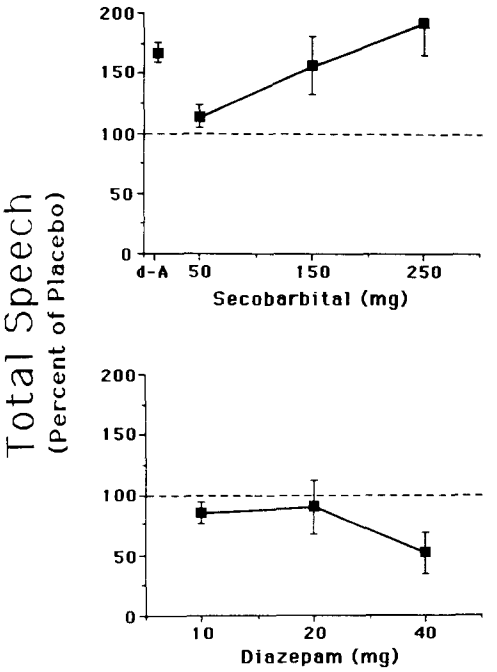

FIG. 1. This figure shows total speech as a function of *d*-amphetamine (d-A) and secobarbital dose in the top panel and diazepam dose in the bottom panel. Total speech is shown as a percent of placebo levels; values above the dashed, horizontal lines represent increases above placebo levels, while values at or below the lines represent no-change and decreases from placebo levels, respectively. Data points are mean values for all subjects in the respective experiments; brackets represent ±1 S.E.M.

Within-session analysis of the distribution of talking across time revealed that seconds of speech decreased significantly across the session ($p<0.02$), but there was not a significant dose × time interaction. For all of the doses, approximately 30% of total talking occurred during the first quarter of the session and the other 70% was equally distributed across the three remaining quarters of the session. Thus, the increases in total speech observed with the 150 and 250 mg doses did not change the within-session distribution of talking.

d-Amphetamine. The 25 mg dose of *d*-amphetamine also increased total talking significantly above placebo levels, which is also shown in the upper panel of Fig. 1 ($p<0.02$). This effect was observed in all six subjects studied. The within-session distribution of talking was similar to the patterns observed with placebo and the active doses of secobarbital.

Diazepam. In contrast to the effects observed with secobarbital and *d*-amphetamine, diazepam generally decreased total speech (Fig. 1, lower panel), although this effect did not reach statistical

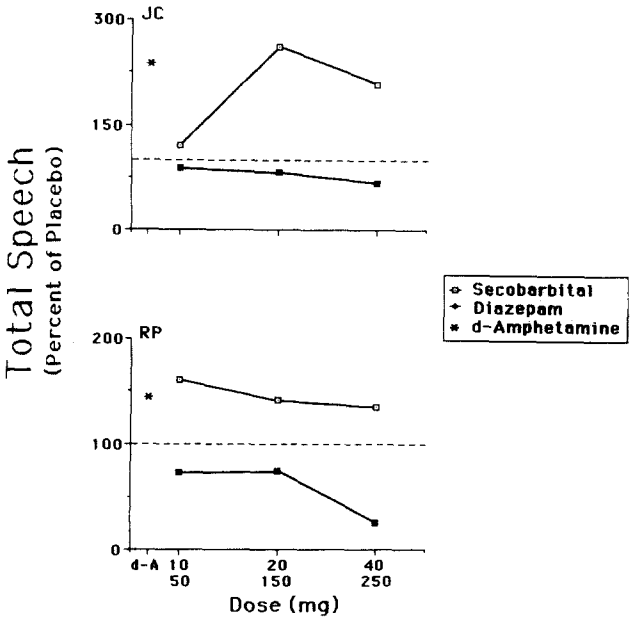

FIG. 2. This figure shows results from two individual subjects who participated in Experiments 1 and 2. Total speech is presented as a percent of control, as in Fig. 1. The *d*-amphetamine (d-A) dose was 25 mg; the secobarbital doses were 50, 150 and 250 mg; the diazepam doses were 10, 20, and 40 mg. Data points represent means of three observations per drug dose.

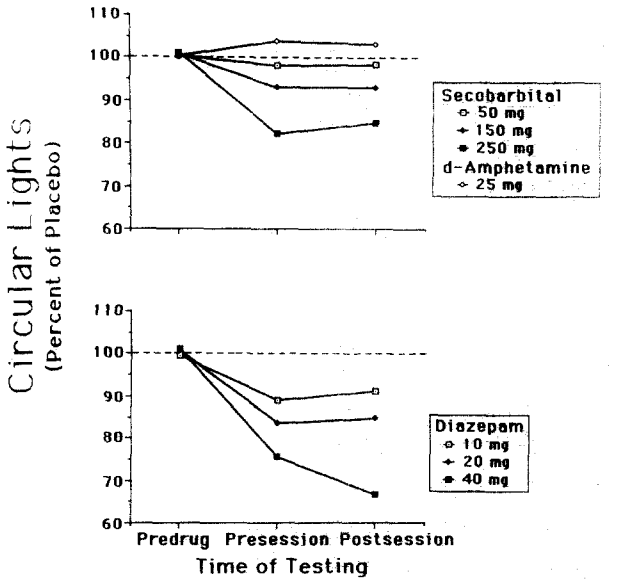

FIG. 3. This figure shows effects on rates of responding in the circular-lights task prior to receiving drug (Predrug), immediately before the talking sessions (Presession), and immediately after the talking sessions (Postsession). Effects of *d*-amphetamine and secobarbital (top panel) and diazepam are shown as a percent of placebo levels. Data points represent mean values for all subjects in the respective experiments.

significance ($p<0.11$). Seven of the nine subjects who received diazepam exhibited decreases in total speech, while two other subjects exhibited increases. Interestingly, diazepam decreased talking in the two subjects who participated in Experiments 1 and 2, whereas secobarbital had increased talking in these two subjects (Fig. 2).

Similar to the within-session patterns observed with secobarbital and *d*-amphetamine, talking decreased significantly as a function of time during the session ($p<0.001$), but there were no significant dose × time interactions. That is, the within-session distribution of talking did not differ between placebo and the active doses of diazepam.

Circular Lights

Secobarbital. There were no significant differences in response rates in the circular-lights procedure across the four dose conditions prior to drug administration. These effects are shown in the upper panel of Fig. 3 with the effects of the active doses represented as percent of placebo values. When measured immediately prior to the talking session, response rates in the circular lights procedure decreased as an orderly function of drug dose and remained below placebo levels during the observation taken immediately after the talking session ($p<0.005$, $p<0.001$, respectively). Prior to the talking session, the rates observed with the 250 mg dose were below those observed with the placebo, 50 and 150 mg doses. All of these differences remained significant after the talking session and the effect of the 150 mg dose was also significant at that time.

d-*Amphetamine.* There was no significant difference between *d*-amphetamine and placebo at predrug. Prior to the talking session, *d*-amphetamine increased response rates above placebo levels ($p<0.02$). This increase was no longer significant at the completion of the talking session. These effects are also shown in the upper panel of Fig. 3.

Diazepam. The effects of diazepam on circular-lights performance are shown in the lower panel of Fig. 3. Rates differed slightly across the dose conditions prior to drug administration with values for the 20 and 40 mg doses being above those observed with placebo and the 10 mg dose ($p<0.05$). Rates decreased as an orderly function of dose immediately prior to and after the talking session ($p<0.0001$ both observations). At both of these postdrug observations, all of the active doses differed significantly from placebo. The 40 mg dose also differed from the 10 mg dose. The 10 and 20 mg doses did not differ significantly prior to or after the talking sessions.

Self-Reports

Secobarbital. The results obtained with secobarbital on the various self-report measures are shown in Table 1. Secobarbital significantly increased visual-analog ratings of drug-produced "high," drug effect and drunkenness ($p<0.001$, $p<0.01$, $p<0.001$, respectively); ratings of sleepiness, relaxed and alert were not significantly affected. On the ARCI subscales, secobarbital significantly decreased scores on the BG (benzedrine) scale and increased scores on the PCAG (sedation) scale ($p<0.05$, $p<0.01$, respectively). There were no other significant effects, although the increasing trend on the MBG scale was marginally significant ($p<0.07$). On the POMS, secobarbital increased scores on the confusion/bewilderment scale ($p<0.05$), but none of the other subscales were significantly affected.

d-*Amphetamine.* The effects of *d*-amphetamine on the self-report measures are also shown in Table 1. Visual-analog ratings of "high" increased significantly above placebo levels when *d*-amphetamine was administered as did ratings of drug effect and alert, while ratings of sleepiness decreased ($p<0.0001$, $p<0.03$, $p<0.01$, $p<0.01$, respectively). On the ARCI, *d*-amphetamine

TABLE 1

SUMMARY OF THE EFFECTS OF *d*-AMPHETAMINE AND SECOBARBITAL ON SELF-REPORT MEASURES[1]

Measures	*d*-Amphetamine 25 mg	Placebo	Secobarbital 50 mg	Secobarbital 150 mg	Secobarbital 250 mg
Visual Analogs:					
"High"	47.7 (12.3)*	19.8 (13.3)	27.0 (18.3)	37.7 (15.4)*	51.2 (14.9)*†‡
Drug Liking	1.9 (0.4)	1.3 (0.2)	1.4 (0.3)	1.7 (0.5)	1.9 (0.6)
Drug Effect	2.9 (0.3)	1.6 (0.4)	2.4 (0.3)	2.5 (0.3)	3.1 (0.4)*
Sleepy	1.1 (0.1)*	2.1 (0.3)	2.3 (0.3)	2.1 (0.4)	2.5 (0.5)
Drunken	1.7 (0.9)	1.2 (0.2)	1.8 (0.5)	1.9 (0.6)	3.0 (0.8)*
Relaxed	2.7 (0.5)	2.3 (0.5)	2.5 (0.3)	2.5 (0.4)	3.3 (0.3)
Alert and Energetic	3.3 (0.3)*	2.0 (0.5)	2.1 (0.4)	2.0 (0.3)	1.8 (0.4)
ARCI Short Form:					
MBG	7.4 (2.2)*	2.0 (0.6)	2.7 (0.7)	4.5 (1.6)	4.8 (2.1)
PCAG	1.5 (0.7)*	5.3 (1.3)	5.9 (1.2)	6.8 (1.5)	9.1 (1.5)*†
LSD	5.1 (1.2)	3.4 (0.5)	3.5 (0.7)	3.5 (0.6)	4.5 (1.2)
A	4.9 (0.9)*	1.9 (0.4)	2.3 (0.6)	3.3 (0.6)	2.7 (0.6)
BG	7.9 (0.9)*	4.9 (0.8)	4.9 (0.9)	5.1 (0.6)	3.2 (0.8)*†‡
POMS:					
Tension	4.7 (1.9)	3.9 (1.0)	3.3 (0.8)	2.9 (0.6)	3.2 (1.0)
Anger	0.1 (0.1)	0.5 (0.4)	0.3 (0.3)	0.1 (0.1)	0.3 (0.3)
Confused	2.1 (0.8)	3.5 (1.0)	3.4 (0.9)	3.7 (1.0)	5.0 (1.2)*†
Depressed	0.5 (0.5)	0.9 (0.9)	0.9 (0.8)	0.3 (0.2)	1.9 (1.4)
Vigor	22.0 (2.4)*	12.4 (2.0)	11.9 (2.2)	10.7 (2.9)	9.8 (3.2)
Fatigue	0.1 (0.1)	4.8 (3.0)	4.7 (2.4)	2.9 (1.4)	3.7 (2.3)
Friendliness	15.7 (5.1)*	12.7 (2.4)	11.6 (1.5)	11.5 (2.2)	11.0 (2.3)
TMD	−11.1 (4.4)*	7.2 (9.4)	2.1 (5.3)	0.9 (5.0)	5.3 (6.5)

[1]Mean values for 6 subjects; values within parentheses are ± 1 S.E.M.
*Value significantly different from placebo.
†Value significantly different from 50 mg.
‡Value significantly different from 150 mg.

increased scores on the A, BG and MBG subscales, and decreased scores on the PCAG subscale ($p<0.02$, $p<0.05$, $p<0.05$, $p<0.03$, respectively). The LSD subscale was not significantly affected. *d*-Amphetamine increased scores on the vigor, friendliness and total mood clusters of the POMS ($p<0.001$, $p<0.01$, $p<0.01$, respectively). The other clusters of the POMS were not significantly affected.

Diazepam. As shown in Table 2, visual-analog ratings of drug-produced "high," drug effect, sleepiness and drunkenness increased, and alert decreased in a dose-related manner ($p<0.0001$, $p<0.002$, $p<0.002$, $p<0.0001$, $p<0.001$, respectively). Ratings of drug-liking and relaxed were not significantly affected. Scores on the ARCI BG (benzedrine) subscale decreased and scores on the PCAG and LSD subscales increased significantly as a function of dose ($p<0.0002$, $p<0.0001$, $p<0.0004$, respectively). The A and MBG subscales were not significantly affected by diazepam. Scores on the POMS vigor and friendliness clusters decreased significantly and scores on fatigue, confusion/bewilderment and the POMS total mood cluster increased as a function of dose ($p<0.01$, $p<0.01$, $p<0.001$, $p<0.004$, $p<0.01$, respectively). The other clusters of the POMS were not significantly affected by diazepam.

EXPERIMENT 3: WITHIN-SUBJECT COMPARISON OF SECOBARBITAL AND DIAZEPAM

For a further comparison of the effects of secobarbital and diazepam on speech, a within-subject assessment of both compounds was investigated in this experiment. A double-blind crossover design was used such that subjects were exposed to all doses of both compounds. Order of exposure to the two compounds was counterbalanced across subjects. A one-week washout period, during which subjects received placebo, was interpolated between testing secobarbital and diazepam.

Subjects

Subjects were four healthy, male volunteers. Their mean age and body weight were 28.3 years (S.D. = 5.5) and 176.5 lbs (S.D. = 34.8), respectively. Medical screening, informed consent, monetary compensation and restrictions on food and drug use were the same as in Experiments 1 and 2.

Drugs

Subjects ingested two size 0 opaque capsules 60 min prior to the start of the talking sessions. Two subjects received diazepam (0, 5, 15, 25 mg) in a mixed order during their first four sessions, followed by two sessions of placebo washout and then four sessions of secobarbital sodium (0, 50, 150, 250 mg) in mixed order; the two other subjects received secobarbital first and diazepam last. A minimum of 72 hr elapsed between drug sessions.

Setting and Apparatus

The setting and apparatus for the talking sessions and circular-

TABLE 2

SUMMARY OF THE EFFECTS OF DIAZEPAM ON SELF-REPORT MEASURES[1]

Measures	Diazepam Dose			
	Placebo	10 mg	20 mg	40 mg
Visual Analogs:				
"High"	19.0 (8.1)	34.8 (7.3)*	45.2 (7.3)*	58.6 (7.7)*†‡
Drug Liking	1.7 (0.4)	1.4 (0.4)	1.6 (0.4)	1.2 (0.5)
Drug effect	1.8 (0.3)	3.0 (0.4)	3.3 (0.3)*	3.5 (0.3)*
Sleepy	1.8 (0.3)	2.7 (0.5)*	3.3 (0.4)*	3.5 (0.3)*
Drunken	1.3 (0.2)	1.9 (0.3)	2.3 (0.4)*	3.0 (0.4)*†
Relaxed	3.2 (0.4)	3.5 (0.3)	3.4 (0.3)	3.1 (0.5)
Alert & Energetic	2.9 (0.4)	2.1 (0.4)	1.8 (0.3)	1.3 (0.2)*
ARCI Short Form:				
MBG	4.7 (1.7)	5.4 (1.8)	3.2 (1.4)	3.7 (1.5)
PCAG	3.9 (1.6)	7.9 (1.8)*	10.9 (1.4)*†	12.2 (0.6)*†
LSD	3.6 (0.6)	3.8 (0.7)	3.8 (0.5)	5.1 (0.6)*†‡
A	3.6 (0.9)	3.4 (0.8)	2.5 (0.5)	2.1 (0.5)
Bg	6.0 (1.2)	3.4 (1.3)*	2.1 (0.8)*	1.2 (0.4)*†
POMS:				
Tension	4.4 (1.4)	3.7 (1.3)	3.9 (1.4)	5.4 (2.7)
Anger	4.0 (3.0)	2.8 (2.6)	3.3 (2.6)	7.6 (4.1)
Confused	3.6 (0.9)	4.7 (1.3)	6.2 (1.5)	10.1 (2.7)*†‡
Depressed	3.7 (2.1)	2.7 (1.6)	3.3 (1.8)	6.9 (4.1)
Vigor	13.9 (3.7)	12.7 (4.2)	8.8 (3.1)*	7.8 (2.3)*
Fatigue	2.6 (1.4)	4.2 (1.8)	8.1 (2.0)*	10.8 (3.2)*†
Friendliness	13.3 (2.2)	12.9 (2.4)	10 (2.3)*	8.9 (1.5)*
TMD	4.6 (7.6)	5.1 (7.1)	15.9 (3.9)	31.5 (13.1)*†

[1]Mean scores for 9 subjects; values within parentheses are ± 1 S.E.M.
*Value significantly different from placebo.
†Value significantly different from 10 mg.
‡Value significantly different from 20 mg.

lights device were the same as in Experiments 1 and 2. Self-report measures were completed on a Commodore 64 microprocessor in this experiment instead of the paper and pencil measures used in Experiments 1 and 2.

Procedure

The talking sessions were conducted as in Experiments 1 and 2, but there were some modifications in the self-report battery. Subjects completed 7 visual-analog scales at predrug, 20, 50 and 100 min postdrug; they rated the strength of the drug effect, whether it had bad effects, whether it had good effects, drunkenness, how alert and energetic they felt, and how sleepy they felt from 0 (not at all) to 100 (extremely). The ARCI short-form was completed at 100-min postdrug; the POMS was omitted. Subjects also completed the Digit Symbol Substitution Test and the Repeated Acquisition of Behavioral Chains procedures at predrug, 20, 50 and 100 min postdrug, but those data are included in another report. The 40-min talking sessions began immediately following the 50-min postdrug observations on these other measures.

Data Analysis

Inferential statistics were not used in analyzing the results from this experiment due to the small sample size. Effects are presented in figures as percent of placebo value to minimize between-subject differences in absolute scores.

RESULTS

Total Speech

The effects of secobarbital and diazepam on total seconds of speech are shown in Fig. 4 for individual subjects with effects of the active doses represented as percent of placebo values. The 150 mg dose of secobarbital increased talking above placebo levels with three of the four subjects. The 250 mg dose of secobarbital increased talking only in subject 1. The 50 mg dose also increased talking with subject 1 and slightly with subject 3, but not with the other two subjects. Diazepam generally had no effect or decreased talking across the four subjects, although there was a slight increase observed at the 5 mg dose with subject 3.

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Circular Lights

The effects of secobarbital and diazepam on circular-lights performance are shown in Fig. 5 as percent of placebo values for

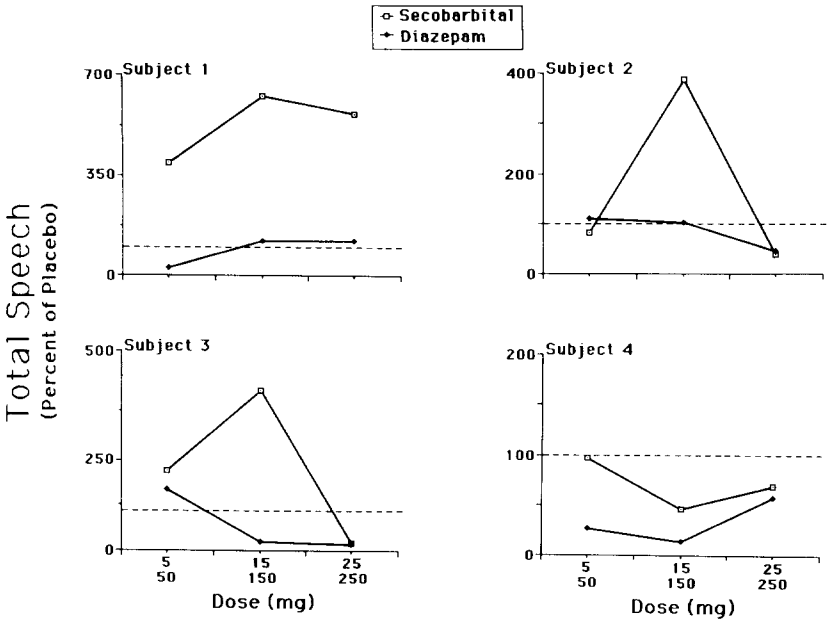

FIG. 4. This figure shows effects on total speech for individual subjects as a function of secobarbital (50, 150, 250 mg) and diazepam (5, 15, 25 mg) dose. Total speech is shown as a percent of placebo levels. Data points represent single observations conducted with each subject at each drug dose.

the four subjects as a group. Both compounds produced orderly dose-related decreases in response rate. Peak effects of secobarbital and diazepam were observed at 100 min and 50 min postdrug, respectively. The magnitude of performance impairment was similar for the two highest drug doses. The highest secobarbital dose (250 mg) produced a 35% percent decrease in response rate, while the highest dose of diazepam (25 mg) produced a 25% decrease in this measure.

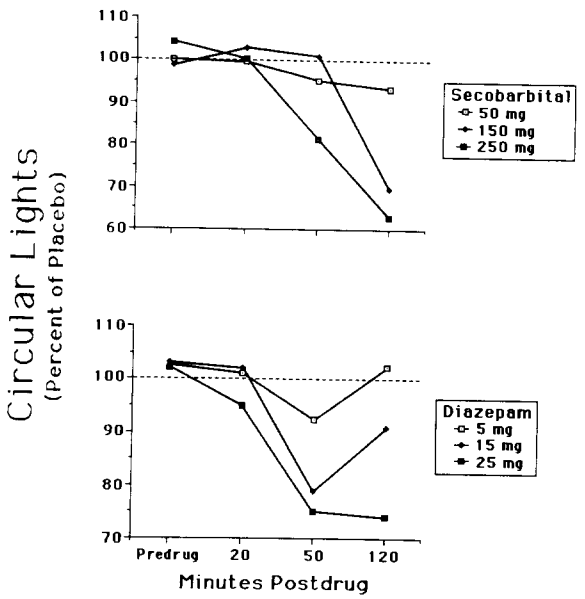

FIG. 5. This figure shows effects of secobarbital and diazepam on rates of responding in the circular-lights task prior to receiving drug (Predrug), 20 minutes postdrug, 50 minutes postdrug (before the talking session), and 100 minutes postdrug (after the talking session). Effects are shown as a percent of placebo levels. Data points represent mean values for four subjects.

Self-Reports

As is shown in Tables 3 and 4, secobarbital and diazepam produced similar effects on the self-report measures. Both compounds produced peak effects on these measures at 100 min postdrug, immediately following the talking session. Both compounds increased ratings on the drug effect, bad effects, good effects, drunken, drug-liking and sleepy scales, and decreased ratings on the alert and energetic scales. On the ARCI, both compounds increased scores on the PCAG, LSD, and MBG subscales, and decreased scores on the BG scale. Secobarbital decreased scores on the A subscale, while diazepam did not; however, the placebo values for diazepam were relatively low on this scale making it difficult to detect a decrease with the active doses of diazepam.

GENERAL DISCUSSION

The purpose of the present study was to investigate the generality of the rate-increasing effects on monologue speech observed with alcohol (15) and *d*-amphetamine (23) to other drugs of abuse. The increases in talking observed with secobarbital in this study demonstrate the generality of this effect to the barbiturates. Diazepam, however, did not reliably increase and sometimes decreased talking. Thus, there also appears to be pharmacological specificity, or limits, to the generality of this effect. The increases in talking observed with *d*-amphetamine in the present study provide a direct replication of our earlier findings (23).

While *d*-amphetamine and secobarbital both increased talking, their effects on rates of responding in the circular-lights procedure

TABLE 3

VISUAL-ANALOG SCORES (PEAK EFFECTS)

	Secobarbital			
	Placebo	50 mg	150 mg	250 mg
Drug Effect	23.5 (6.7)	32.8 (11.3)	59.5 (13.9)	76.0 (7.8)
Bad Effects	18.5 (10.4)	17.5 (8.8)	25.5 (10.6)	38.5 (20.7)
Good Effects	11.8 (3.1)	23.8 (13.2)	47.0 (15.7)	35.3 (11.6)
Drunken	1.3 (0.6)	5.5 (4.2)	25.5 (7.0)	31.0 (18.5)
Alert and Energetic	58.3 (16.8)	50.0 (12.4)	40.5 (13.4)	34.8 (17.2)
Sleepy	2.3 (0.5)	27.0 (17.7)	32.5 (7.8)	60.5 (14.0)
Drug Liking	29.8 (12.8)	26.0 (14.2)	54.5 (17.9)	37.8 (13.6)
	Diazepam			
	Placebo	5 mg	15 mg	25 mg
Drug Effect	12.3 (5.7)	34.0 (7.3)	53.0 (5.5)	51.5 (8.9)
Bad Effects	3.8 (1.9)	18.8 (9.3)	33.3 (17.5)	36.3 (18.1)
Good Effects	9.8 (7.8)	52.3 (18.9)	19.0 (13.2)	32.5 (11.1)
Drunken	1.8 (0.9)	14.5 (9)	26.3 (8.8)	46.5 (14.6)
Alert and Energetic	77.8 (12.0)	53.8 (10.8)	34.0 (18.7)	28.5 (16.2)
Sleepy	5.5 (2.7)	31.5 (3.9)	45.3 (18.0)	59.3 (18.5)
Drug Liking	32.3 (11.2)	44.8 (9.0)	19.0 (14.1)	42.3 (14.0)

*Mean scores for 4 subjects; values within parentheses are ± 1 S.E.M.

and self-reports differed. *d*-Amphetamine produced a profile of effects characteristic of a prototypical CNS stimulant, with increases in rates of responding in the circular-lights procedure, and increases in the ARCI "euphoria" (MBG) and stimulant (A, BG) subscales and ratings of vigor, friendliness, etc. [e.g., (6, 14, 23)]. Secobarbital, in contrast, generally produced effects characteristic of a prototypical CNS depressant, with decreases in rates of responding in the circular-lights procedure, increases in the ARCI sedation subscale (PCAG), and increases in ratings of drunkenness, confusion, etc. [e.g., (10,21)]. Thus, the common effect these two compounds produce on talking is a selective behavioral effect.

The difference between the effects of secobarbital and diazepam on talking was unexpected since the behavioral pharmacology of the barbiturates and benzodiazepines are often similar. For example, their effects in studies with nonhumans responding under various schedules of positive and negative reinforcement are typically the same, as are their effects on shock-suppressed responding (17). However, a careful review of the literature on the behavioral effects of these compounds also reveals many instances in which their effects differ. Ator and Griffiths (3), for example, have demonstrated that baboons and rats trained to discriminate lorazepam from saline do not generalize to pentobarbital. File (5) has reported that the effects of pentobarbital and diazepam can be differentiated in a nonhuman social interaction paradigm. The effects of barbiturates and benzodiazepines on visual tracking can be differentiated in nonhumans (1) and humans (16). The barbiturates generally maintain higher rates of drug ingestion than the benzodiazepines in nonhuman self-administration paradigms [see (2, 11, 24) for reviews]. In human sedative abusers, pentobarbital maintains higher rates of self-administration than diazepam (8) and is also chosen over diazepam in an exclusive choice paradigm (9). Chronic administration of diazepam to sedative abusers has been reported to increase staff ratings of subjects' hostility and complaining, whereas administration of pentobarbital did not (10). Additionally, sedative abusers have been reported to underestimate their level of drug-induced behavioral impairment while under the influence of triazolam, but not pentobarbital (19). It is noteworthy that the differences observed between the barbiturates and benzodiazepines in sedative abusers with regard to self-administration, hostility, and estimations of drug impairment occurred at doses that produced similar effects on other measures, e.g., staff ratings of overall drug effect, amount of daytime sleeping, and performance on behavioral tasks.

One possible explanation for why secobarbital and diazepam differed in their effects on talking in the present study is that the diazepam doses we used were relatively high, and too sedating, to increase talking. For example, the 40 mg dose of diazepam used in Experiment 1 produced a larger decrease in response rates in the circular-lights procedure than did the highest dose (250) of secobarbital. Additionally, the 20 and 40 mg doses of diazepam also significantly decreased the friendliness and vigor clusters and

TABLE 4

ADDICTION RESEARCH CENTER INVENTORY*

	PCAG	BG	A	LSD	MBG
			Secobarbital		
Placebo	2.8 (0.8)	8.0 (1.5)	3.8 (1.1)	3.8 (1.0)	3.8 (1.8)
50 mg	6.8 (1.3)	5.5 (0.3)	3.3 (1.7)	3.0 (0.7)	4.8 (1.8)
150 mg	7.3 (2.2)	5.8 (1.0)	4.5 (1.7)	4.3 (0.7)	5.5 (2.6)
250 mg	10.0 (1.4)	3.2 (0.8)	2.4 (0.3)	5.6 (1.5)	5.0 (1.2)
			Diazepam		
Placebo	4.3 (1.9)	6.0 (0.6)	2.8 (0.8)	4.8 (0.8)	3.5 (1.2)
5 mg	6.8 (1.3)	4.8 (1.0)	2.5 (0.9)	4.3 (1.0)	2.5 (1.2)
15 mg	7.8 (1.7)	3.8 (1.8)	2.8 (1.4)	3.0 (1.1)	4.0 (2.7)
25 mg	10.5 (1.6)	2.5 (0.9)	2.8 (0.5)	5.3 (0.3)	4.8 (0.6)

*Mean scores for four subjects; values within parentheses are ± 1 S.E.M.

increased the fatigue clusters of the POMS in that experiment, while none of the secobarbital doses did so. While these observations appear to support a position that the differences between diazepam and secobarbital can be accounted for on the basis of sedation, other observations from the circular-lights and self-report forms do not support that position. For example, the 10 mg dose of diazepam produced effects on circular-lights and the self-report measures that were similar to those of the 150 mg dose of secobarbital, yet the 10 mg dose of diazepam did not increase talking. Additionally, we used a lower range of diazepam doses in Experiment 3 that produced reasonably comparable effects to those produced by secobarbital on the circular-lights and self-report measures, yet diazepam still failed to increase talking. Thus, neither effects on the circular-lights or self-report measures were reliable predictors of effects on talking. In our opinion, the differences we observed between the effects of diazepam and secobarbital on talking do not appear to be well accounted for on the basis of dose selection or sedation.

Before being tested in the monologue speaking procedure, *d*-amphetamine and secobarbital had been demonstrated to increase rates of dyadic social conversation (6,21). To our knowledge, the effects of diazepam on social conversation have not been reported. It will be important to determine if the differences observed between secobarbital and diazepam in the monologue speaking procedure also occur in social arrangements.

As was noted in our prior report on the effects of alcohol on monologue speech (15), the observation of drug produced increases in speech in the absence of a social partner raises interesting questions about the significance and specificity of the effects that have been observed in social contexts. Perhaps the increases in social conversation observed with *d*-amphetamine, alcohol, and secobarbital in prior studies were due to general increases in talkativeness rather than a specific effect on social interaction. A social context does not appear to be a necessary condition for observing characteristic drug-produced increases in speech quantity. However, the content of the speech monologues produced by subjects in these isolated speech experiments are similar to naturalistic conversational speech. Subjects often talked about everyday events in their lives. Thus, there may be something about naturalistic speech and the variables that control it, historically or presently, that accounts for the similar drug effects observed in the dyadic and monologue talking arrangements.

Overall, the present study demonstrates that the increases in monologue talking observed previously with *d*-amphetamine and alcohol also occur with secobarbital. Pharmacological and behavioral specificity was demonstrated since diazepam failed to reliably increase talking, and increases in talking were not predictable from effects on the circular-lights and self-report measures. Whether the differences observed between secobarbital and diazepam in the monologue talking arrangement used in this study will also occur in social contexts is an important matter for future investigation. Future studies are also needed to identify similarities and differences in the effects of drugs on social conversation versus other types of verbal and nonverbal behavior, to clarify the role of social stimuli in determining drug effects on talking, and to determine whether drugs have specific effects on social behavior or the behavioral effects of social stimuli.

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