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Sentence Memory Affected by Vasopressin Analog (DDAVP) in Cross-Over Experiment

ROBERT E. TILL AND BILL E. BECKWITH[1]

Department of Psychology, University of North Dakota
Box 7187, University Station, Grand Forks, ND 58202

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TILL, R. E. AND B. E. BECKWITH. *Sentence memory affected by vasopressin analog (DDAVP) in cross-over experiment.* PEPTIDES **6**(3) 397–402, 1985.—DDAVP has been shown to facilitate memory, especially retrieval, in humans. Healthy young male adult subjects received DDAVP (60 μg) in a cross-over design with a one-week interval between sessions. Results indicated that DDAVP improved immediate memory during the first but not the second testing session, particularly for low-verbal subjects. Treatment with DDAVP also facilitated delayed (one-week) recall in the opposite group, a cross-over interaction that suggests a retrieval locus for the DDAVP effect. Furthermore, since DDAVP improved immediate memory more for low-verbal subjects and delayed memory more for high-verbal subjects, it appears that individual difference factors will be important in understanding the effects of vasopressin on memory.

DDAVP Memory Neuropeptide Vasopressin

ALTHOUGH vasopressin is probably best known for its function in controlling fluid balance [13], a number of recent studies, using both human and nonhuman subjects, have suggested that it may facilitate learning and memory [3, 4, 7, 10, 11]. Furthermore, recent work with humans has also begun to demonstrate improvement in learning and memory situations when a particular synthetic analog of vasopressin is used, namely, l-desamino-8-D-arginine vasopressin (DDAVP).

For example, Weingartner *et al.* [11] tested healthy and cognitively impaired adults who had been treated with DDAVP for a period of two to three weeks and found evidence of increased completeness, organization, and consistency of recall. While the sample sizes were small (e.g., six healthy volunteers), improvement in performance was reported to be in contrast to minimal changes in the placebo group.

Using larger samples, Beckwith and his colleagues [1, 2, 3] have found improved performance even with a single, acute dose of DDAVP administered intranasally. The improvement, so far reported only for male adults, was seen in a concept learning task, a visual reaction time task, and an auditory sentence memory task. Two of these studies [1,2] suggested that DDAVP enhances performance by improving attentional processes (e.g., increased alertness, faster stimulus encoding) rather than memory processes (e.g., faster search of elements in a memory set). Thus, Beckwith *et al.* [2] speculated that earlier studies claiming to show memory effects of vasopressin might actually reflect the peptide's general enhancement of encoding processes. The third study [3], which included various encoding and retrieval conditions, also found a general improvement for males tested with DDAVP and no interactions, despite the inclusion of relatively "matched" and "mismatched" combinations of encoding and retrieval tasks. That is, treatment with DDAVP led to better performance when encoding and retrieval were optimally matched (e.g., comprehension, cued recall) as well as when they were mismatched (e.g., comprehension, free recall). In sum, the Beckwith *et al.* studies show results consistent with some kind of attention hypothesis of vasopressin's effect.

Even if vasopressin's effect is the result of improved attention, little can be said about the mechanism by which it influences behavior. According to one view [7], vasopressin influences behavior through its peripheral autonomic effects that alter arousal. Although change in arousal might predict general improvement on memory measures, Beckwith *et al.* [1,2] have found none of the corresponding changes in blood pressure or heart rate that might be expected with increased arousal. Thus, a simple peripheral interpretation of DDAVP effects on performance seems unlikely. An alternative view, based on central nervous system effects, remains a possibility.

Regardless of the mechanism by which vasopressin affects behavior, it is not yet clear whether such effects operate at encoding or retrieval stages of memory. If DDAVP is administered so as to selectively influence encoding or retrieval processes, its effect might be "attentional" yet localized in encoding or retrieval activities. That is, DDAVP might improve memory because information is better encoded at study (quantitatively or qualitatively) or because it is better retrieved at test (more of it or more accurately). The previous studies of vasopressin's effect have not addressed this issue of locus.

[1]Requests for reprints should be addressed to Bill E. Beckwith, Department of Psychology, University of North Dakota, Box 7187, University Station, Grand Forks, ND 58202.

The present study sought first to replicate the finding of improvement with DDAVP through the use of a repeated-measures, "cross-over" design in which the DDAVP group would later be tested under placebo treatment and the placebo group would later receive the DDAVP treatment. Ideally, a cross-over interaction would be found in which the Vasopressin-Placebo group would perform better on material presented and tested during the first session but worse on material presented and tested during the second session, as compared to the Placebo-Vasopressin group. That is, at each test session, we expected the group encoding and retrieving under DDAVP treatment to recall more than the placebo group.

A second goal of the study was separation of the DDAVP effect on encoding and retrieval. At the end of the second session, individuals could be asked for delayed recall of the first session's material. If the effect of DDAVP is due to improvement at encoding, the Vasopressin-Pacebo group would be expected to recall more than the Placebo-Vasopressin group at delayed recall and at immediate recall. In contrast, if the effect of DDAVP is due to improvement at retireval, there should be a cross-over interaction such that the group under DDAVP treatment at test time recalls more (i.e., Vasopressin-Placebo group better at immediate test, Placebo-Vasopressin group better at delayed test).

The study also included two additional variables known to influence performance in a sentence memory task [5, 8, 9]: scoring criterion and vocabulary level. No specific predictions were made for the scoring variable, except that recall would be higher under lenient (gist) scoring than under strict scoring. A vocabulary-level factor was created by splitting each group into high-verbal and low-verbal subgroups since the earlier study by Beckwith *et al.* [3] led us to suspect that improvement with DDAVP might be greater for low-verbal individuals. Thus, we expected vocabulary × treatment interactions.

Finally, a Placebo-Placebo group was added (toward the end of the study) to provide a direct look at practice effects in the absence of DDAVP. Some improvement from the first to the second session was expected.

METHOD

Subjects

Three groups of healthy males, with 14 per group, were included in this study. All were native speakers of English. All reported being free of medication, illness, hypertension (i.e., blood pressure above 140/90 mm of Hg), cardiovascular and renal disease. All reported that they had a normal night's sleep before the day of the experiment, that they had no food since 1300 hr, and that they had had no alcohol or other drugs within 24 hr. All were tested between 1500 and 1900 hr.

All subjects were undergraduates in psychology courses at the University of North Dakota and received course credit for their participation. Procedures had been reviewed and approved by the university's review board. Subjects provided written, informed consent, and retained the right to withdraw from the study at any time.

Subjects' ages ranged from 18 to 33 years, with no significant difference between the three groups. Similarly, there was no difference among the three groups in verbal ability (as measured by a vocabulary test). It should be noted, however, that the Placebo-Placebo group was tested after the other two. Since there are often subtle differences in volunteers across academic semesters, the Placebo-Placebo data are reported separately.

Materials

Two lists of sentences were constructed from materials previously used by Till and his colleagues [7,8]. Each list contained 16 sentences worded such that certain implications would have a high probability during comprehension. Examples are: (1) *The chauffeur drove on the left side,* and (2) *The pupil positioned the thumbtack on the chair.* Average sentence length was approximately 6.0 words for each list. Two randomized sentence orders were constructed, and during sentence presentation half of the subjects in a particular condition heard one while the other half heard the other. Sentences were prerecorded at the rate of one new sentence every 20 sec. Each sentence was read and immediately repeated to minimize perceptual errors. Recorded materials were presented through binaural earphones from a cassette tape.

Two lists of recall cues, one for each sentence, were also constructed. Each cue was a noun referring to information that might be inferred from the sentence, such as an object involved in the described event or the social significance of the event. Cues for the example sentences above are: (1) *England* and (2) *prank.* In contrast to our earlier study [3], cue words were always presented to subjects as a list printed on a single page.

Design and Procedure

Our initial design called for a cross-over experiment in which one group received vasopressin at the first session and a placebo at the second while the other group first received the placebo and then later received vasopressin. Near the end of the data collection, the Placebo-Placebo group was added to the study to clarify practice effects. In all groups, the first and second sessions were separated by about seven days (exceptions to this, 6 days or 8 days, were few and were not peculiar to any of the three groups).

Each subject was tested individually. Upon arrival, health and biographical data were reviewed and blood pressure was recorded. Those subjects with blood pressures above 140/90 mm of Hg were excluded from the study. Each subject took a written vocabulary test (second half of the subtest from the Wechsler Adult Intelligence Scale). Next the individual was randomly assigned to a treatment-order condition. For all subjects at all times, treatment was administered using a double-blind technique.

Treatment consisted of a single administration of either 60 μg of DDAVP in 0.6 ml of solution or 0.6 ml of saline. The fluids were instilled slowly (within 15 sec) into one nostril while the subject reclined on a couch. The individual remained supine for the next 20 min to allow for absorption of the fluid and then was taken to another room for the memory testing.

Subjects were given intentional learning (i.e., "memorize") instructions to carry out while listening to the list of sentences. They made no written responses. Following this task and a one-minute counting task (to minimize short-term memory or recency effects in free recall), a free-recall test was given. Subjects were allowed 5 min to write as many sentences as they could remember, in any order. Exact wording was encouraged but not required.

Individuals returned the following week for the second session and were administered the other treatment, except in

the Placebo-Placebo group which received the placebo on both occasions. The procedure was the same as during the week before. Again, double-blind technique was used. Subjects hearing one sentence list during the first session heard the other list at the second session.

During the second session, additional tests were given following free recall of the second session list. First, a list of cue words for the second session sentences was presented on a sheet of paper and subjects were allowed 5 min to use the cues to help recall. They were told they could write even those words just given in free recall, but of course they were not allowed to look at the free recall sheet. Subjects simply wrote a recalled sentence alongside the cue that reminded them of it.

Second, a delayed recall test was given for the previous week's sentence list. Subjects were allowed 5 min to recall sentences from the previous week.

Finally, at the end of this second session, subjects in all conditions were told that many of them had received vasopressin on one occasion and a placebo on the other. They were asked to indicate whether they thought they had been given vasopressin at this final session. The individual's response from a 6-point scale ("1"=sure no; "6"=sure yes) was recorded.

Scoring

Sentence recall was scored according to strict and lenient criteria used by Till *et al.* [8]. Under strict scoring, only minor grammatical changes and close synonyms were considered acceptable changes of wording. Under lenient scoring, sentences recalled verbatim as well as those containing some substitutions or omissions were considered acceptable. For example, sentences with omissions or redundant information were accepted as long as the agent, verb, and some of the additional information were specified. In contrast to our earlier study [3], acceptable sentences in cued recall were counted regardless of whether they were "correctly" paired with the experimenters' cues.

General criteria for acceptability were studied by two judges who subsequently worked independently, and without awareness of treatment condition, to provide recall scores for each subject. For each individual subject, there were strict and lenient scores for first session recall, second session recall, cued recall, and delayed recall. Over all these measures, for the three groups, the interrater reliability coefficient for the two judges' scores was 0.95 on the average (ranging from 0.88 to 1.00). Scoring discrepancies were resolved conservatively to provide a single set of scores for analysis, i.e., only responses acceptable to both judges were included.

RESULTS

As a check on our double-blind procedure, we first examined subjects' confidence that they had received vasopressin at the second session. An analysis of variance indicated that mean confidence ratings were near the midpoint of the scale and did not differ ($F<1$) for the three groups: Placebo-Vasopressin group ($M=3.64$; $SD=1.34$), Vasopressin-Placebo group ($M=3.21$; $SD=1.37$), and Placebo-Placebo group ($M=3.36$; $SD=1.04$). Interestingly, over the 42 subjects, there was a negative correlation between first session free recall and confidence rating, $r=-.30$, $p<0.05$ (under strict recall scoring). To some extent, those who had low recall scores in the first session were more confident that they had received vasopressin in the second session. Those with high recall scores in the first session were less sure they had been given vasopressin in the second session. Thus, the double-blind procedure seems to have been effective. Judgments about treatment condition appear to be more related to perceived recall performance than to any (subtle) cues about the treatment administered.

TABLE 1

MEANS AND STANDARD DEVIATIONS FOR VOCABULARY SCORES OF LOW-VERBAL AND HIGH-VERBAL SUBJECTS IN EACH TREATMENT-ORDER GROUP

Treatment Group and Measure	Low-Verbal	High-Verbal
Placebo-Vasopressin		
M	15.3	23.4
SD	4.1	4.3
Vasopressin-Placebo		
M	14.9	23.7
SD	3.4	4.0
Placebo-Placebo		
M	14.3	23.7
SD	2.2	3.7

Maximum score=40.
For each mean, n=7.

As noted earlier, the three groups did not differ in age or in vocabulary scores. However, pilot work suggested possible differences in treatment effectiveness for individuals of high and low verbal ability. Thus, we introduced a vocabulary-level factor in our analyses of recall. The split into high- and low-verbal ability had a similar effect on all three groups; an analysis of variance on vocabulary scores showed only a main effect of level, $F(1,36)=60.3$, $p<0.001$. Means and standard deviations are shown in Table 1.

Placebo-Placebo Control Group

The Placebo-Placebo group was not strictly comparable to the other treatment groups since the subjects were tested at a different time of year. Thus, results were analyzed in a separate (vocabulary level × session × scoring) analysis of variance. As expected, there was a main effect of scoring, $F(1,12)=14.5$, $p<0.01$; recall was greater under lenient scoring than under strict scoring. There was also a main effect of vocabulary level, $F(1,12)=5.33$, $p<0.05$; those with high-verbal ability recalled more than those with low ability. There was no main effect of session and only a marginal interaction of vocabulary level × session, $F(1,12)=3.22$, $p<0.10$. As seen in Fig. 1, there is some suggestion of a session effect (i.e., "practice effect") for low-verbal subjects, but no such trend for high-verbal subjects. Data in Fig. 1 are averaged for the two scoring levels.

Cross-Over Treatment Groups

Recall scores for the Placebo-Vasopressin group and the Vasopressin-Placebo group were analyzed in a four-factor analysis of variance (treatment order × vocabulary × session × scoring). Mean recall is shown in Fig. 1. There was a

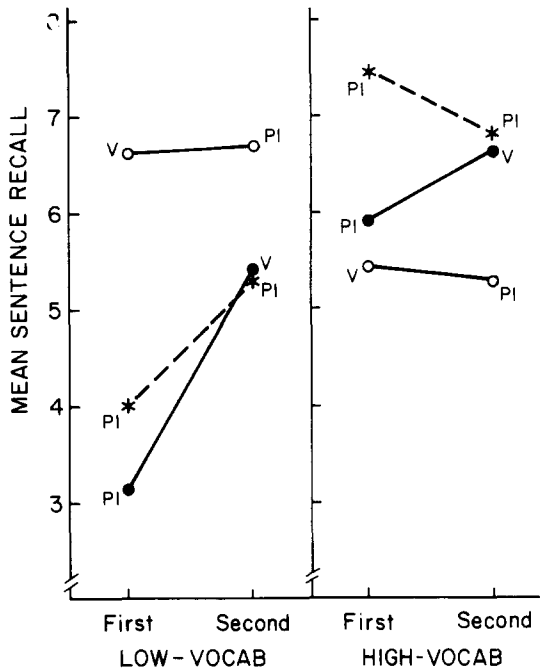

FIG. 1. Mean number of sentences recalled by low-vocabulary and by high-vocabulary subjects treated with vasopressin (V) and placebo (Pl) and tested for immediate recall during first and second sessions. Data for all treatment-order groups are averaged across type of scoring.

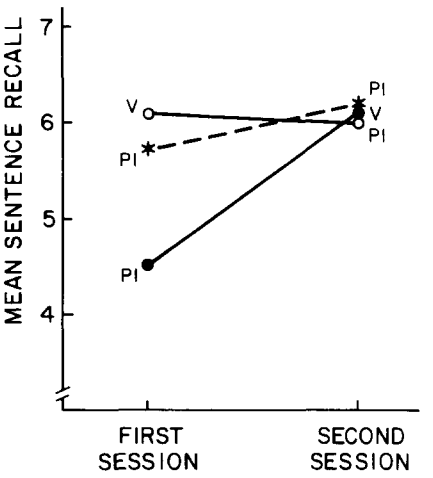

FIG. 2. Mean number of sentences recalled by subjects in the three treatment-order groups: vasopressin (V)—placebo (Pl), placebo—vasopressin, and placebo—placebo. Data are averaged across verbal ability and type of scoring.

main effect of scoring, $F(1,24)=31.9$, $p<0.001$; lenient recall scores were greater than strict recall scores. The treatment order × vocabulary level interaction was significant, $F(1,24)=9.14$, $p<0.01$. Since this interaction is seen when recall is collapsed over session, and therefore over the two kinds of treatment, the result is puzzling and suggests differences between groups randomly assigned to the two treatment orders. On close examination, we found that overall recall of high-verbal subjects in the Placebo-Vasopressin order was not greater than that of high-verbal subjects in the Vasopressin-Placebo order, but overall recall did differ for low-verbal subjects in the two treatment orders, $t(12)=2.87$, $p<0.02$. Thus, low-verbal subjects in the Vasopressin-Placebo group may have been superior to those in the Placebo-Vasopressin group despite our efforts to assign subjects arbitrarily (but keep groups comparable in verbal ability). Alternativley, one would have to argue for carry-over effects of an acute dose of DDAVP influencing performance of the Vasopressin-Placebo group even one week after administration. Finally, the treatment order × session interaction approached significance, $F(1,24)=3.21$, $p<0.09$, reflecting (though weakly) the predicted cross-over interaction. As seen in Fig. 2, with recall collapsed over scoring and vocabulary levels, those treated with DDAVP in the first session outperformed those treated with the placebo, one-tailed $t(26)=1.84$, $p<0.05$. During the second session, however, these same participants (now under treatment with placebo) performed no differently from or only slightly worse than those in the other treatment group. In sum, the treatment order × session interaction was less clearly a "cross-over" interaction than expected, whether because of the general superiority of one group, DDAVP carry-over effects, or other factors.

After second-session recall, subjects were tested for "delayed" recall of the previous week's sentences. Acknowledging that delayed recall was also repeated recall for the first list, we examined recall as a function of time (immediate vs. delayed) in the two treatment-order groups. Recall was analyzed in an analysis of variance (treatment order × time × vocabulary level × scoring). Mean recall for the two groups (as well as for the Placebo-Placebo group) is shown in Figs. 3 and 4.

The analysis indicated a main effect of scoring, $F(1,24)=22.5$, $p<0.001$; lenient scores were greater than strict scores. There was also a time × scoring interaction, $F(1,24)=8.39$, $p<0.01$, reflecting the fact that scoring level had a larger effect on immediate recall (lenient $M=5.79$ vs. strict $M=4.79$) than on delayed recall (lenient $M=1.25$ vs. strict $M=0.96$). More importantly, there was a main effect of time, $F(1,24)=193.5$, $p<0.001$, a treatment order × time interaction, $F(1,24)=14.5$, $p<0.001$, a treatment order × vocabulary level interaction, $F(1,24)=5.27$, $p<0.04$, and a three-way interaction of treatment order × vocabulary level × time, $F(1,24)=6.23$, $p<0.02$. As suggested by Fig. 3, clear treatment effects occurred for low-verbal subjects at immediate testing, $t(12)=3.05$, $p<0.01$, and for high-verbal subjects at delayed testing, $t(12)=1.83$, $p<0.05$ (one-tailed). Alternatively, Fig. 4, which collapses over vocabulary levels, shows treatment effects at immediate and delayed tests that were significant, by one-tailed t-tests, at $p<0.06$ or better. Thus, this immediate vs. delayed recall analysis revealed a stronger "cross-over" pattern than the preceding analysis.

Immediate and delayed recall for the Placebo-Placebo group is also shown in Figs. 3 and 4 (unconnected data points), although the data were not included in the analysis of variance. As noted earlier, this control group was tested at a later time than the other two treatment-order groups.

The cued recall data were gathered only after second-session recall, for exploratory purposes. A treatment order × vocabulary level × scoring analysis of variance indicated only a main effect of scoring, $F(1,24)=27.3$, $p<0.001$; lenient scores were higher than strict scores. In view of the procedural changes from the Beckwith *et al.* [3] study, this outcome is not surprising. In contrast to the earlier study,

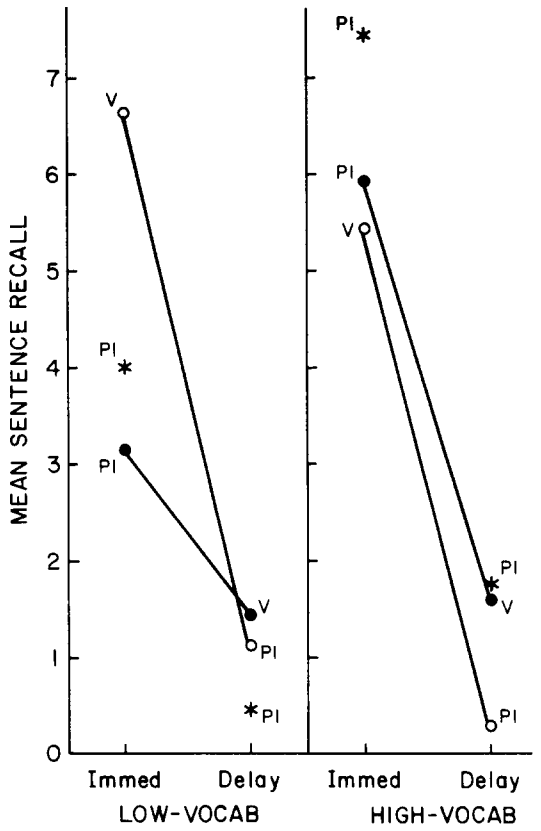

FIG. 3. Mean number of sentences recalled by low-vocabulary and by high-vocabulary subjects treated with vasopressin (V) and placebo (Pl) and tested for immediate and delayed recall of first-session material. Data are averaged across type of scoring.

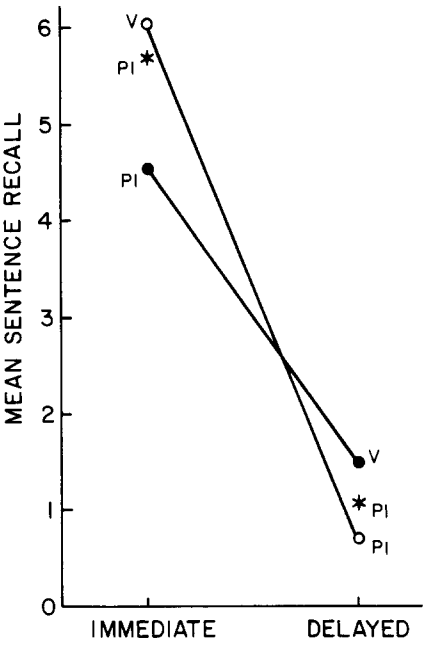

FIG. 4. Mean number of sentences recalled, during immediate and delayed tests of first-session material, by subjects in the three treatment-order groups: vasopressin (V)—placebo (Pl), placebo—vasopressin, and placebo—placebo. Data are averaged across verbal ability and type of scoring.

cued recall here (a) occurred at a second rather than a first testing, (b) was always "piggybacked" on free recall, (c) utilized written rather than auditory cues, (d) was self-paced, and (e) was scored without regard to "correct" pairing of sentences with the cues. No further analysis is given here.

DISCUSSION

The present results provide evidence that DDAVP may facilitate memory, particularly retrieval processes. The effect was clearest for individuals treated with DDAVP, rather than placebo, during the first test session. During a second session one week later, in which new material was presented and tested, the two treatment groups showed little or no difference in recall. During this second session, however, the groups *did* differ in their (delayed) recall of the previous week's material, which argues against a simple superiority or carry-over interpretation of the Vasopressin-Placebo group's immediate recall of the two lists.

Although a practice effect (improvement from first to second session) might explain the absence of a stronger cross-over interaction in Fig. 2, the direct evidence for a practice effect (in the Placebo-Placebo group) is weak. Alternatively, it might be argued that the Vasopressin-Placebo group still benefitted during the second session from the acute treatment with DDAVP the previous week. Thus, both groups would have similar recall during the second session because their treatment status was functionally the same (i.e., DDAVP). As noted, this "carry-over" interpretation does not account for the cross-over interaction seen in Fig. 4. There the DDAVP advantage appeared at both immediate and delayed recall and was of similar significance. Rather than a carry-over effect, Fig. 4 suggests that what is critical is the treatment operative at the time of retrieval.

Considering the data of Figs. 2 and 4, we suggest that there may be improvement of learning (encoding) with practice and that the DDAVP advantage may disappear quickly with new but similar lists. At the same time, there may be a general DDAVP advantage in retrieving stored information, particularly old information. Studies using several lists and several testings will be needed to clarify these issues about the locus of the DDAVP effects (e.g., successive tests of both immediate and delayed recall).

The effects of verbal ability qualify to some extent the patterns described above and suggest that the DDAVP advantage is mainly seen in the low-verbal subjects. In Fig. 1, for example, all seven low-verbal subjects in the Placebo-Vasopressin subgroup showed an increase in recall from the first to the second session ($p<0.02$, sign test). No other subgroup showed a significant change (by sign test) from the first to the second session. In Fig. 3 it can be seen that the low verbal subgroups differ greatly in immediate recall while performance seems equally poor at delayed recall. DDAVP may be influencing encoding more than retrieval processes, for these subgroups. In contrast, the high-verbal subgroups were quite similar on immediate recall, but differed on delayed recall. Perhaps these individuals, already quite proficient in verbal learning tasks, are helped by DDAVP only in the retrieval process. Though puzzling, such interactions are increasingly reported in the memory literature [5,6] and indi-

cate the importance of studying participant characteristics in relation to experimental variables.

Several findings converge to suggest a connection between the DDAVP effect and task proficiency, at least for verbal memory tasks. The present data show a treatment effect at the first session that disappears with a new list at the second session. The present study also shows a stronger treatment effect for low-verbal than for high-verbal individuals. The Beckwith *et al.* [3] finding of a significant DDAVP effect in males, but not in females, is also consistent with the pattern (in view of the literature on sex differences suggesting that males have lower verbal proficiency). In sum, these findings suggest a remedial function; DDAVP appears to help individuals who are disadvantaged with respect to the test situation.

The present study is a start in trying to localize DDAVP effects. It raises new questions about subject characteristics (high-verbal vs. low-verbal) and treatment locus (encoding vs. retrieval), but it also helps eliminate some rival explanations of DDAVP effects. For example, with the groups equated on vocabulary and age, we were confident that the treatment effects were not confounded with simple verbal ability differences or age differences of the groups. Also, subjects' confidence ratings of their treatment condition provided assurance that the double-blind procedure was effective. Finally, the use of a repeated-measures design allowed a check on practice effects and change within individuals. Thus, the present study rules out certain artifactual explanations and sets the stage for more sophisticated studies of the locus of the DDAVP effect.

At another level, the question of the locus of vasopressin's effect on memory must also lead to a physiological mechanism. There seem to be two general views of how vasopressin influences behavior and they need not be mutually exclusive. On the one hand, vasopressin may influence behavior through peripheral autonomic effects that alter arousal. However, DDAVP is an analog that has been synthesized to be devoid of pressor action [13], which of course argues against a peripheral explanation of the present data. The simple peripheral account [7] also cannot explain interactions of DDAVP treatment with verbal ability. An alternative view, that vasopressin enhances learning and memory by means of a central nervous system effect [4,10], is more attractive though not yet established.

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