Table 1. Means and standard deviations of the learning rate scores for the drug conditions.

| Dose (mg) | Mean | Standard deviation |
|---|---|---|
| | *Placebo* | |
| | .275 | .084 |
| | *Magnesium pemoline* | |
| 6.25 | .333 | .045 |
| 12.5 | .356 | .077 |
| 25.0 | .490 | .077 |
| | *Dextroamphetamine* | |
| 15.0 | .506 | .055 |

curves for differences in learning rate during the period of greatest growth, it was necessary to estimate this segment of the curve. The learning curve was averaged for all subjects and each trial, beginning with trial 14, was compared with the last trial with Dunnett's test against a control (*8*). The first trial significantly different ($t = 2.74$, $df$ 14/350, $P<.05$, one-tailed test) from trial 15 (asymptote) was trial 8. Consequently, the period of greatest learning was indexed by the average of the proportions of correct responses over trials 2 through 9. This value was used to calculate the learning rate.

A significant difference in the average value of this score indicates that the functions which relate practice to learning differ. For the various drug conditions Fig. 1 shows the learning curves and Table 1 shows the means of the learning rate scores (smaller values indicate more rapid improvement). Mean learning rate was fastest under placebo and increasingly slower under 6.25 mg of magnesium pemoline, 12.5 mg of magnesium pemoline, 25 mg of magnesium pemoline, and 15 mg of dextroamphetamine. Each active drug condition was compared with the placebo condition, with Dunnett's test against a control. Dextroamphetamine was the only drug condition significantly different from placebo ($t = 2.31$, $df$ 5/25, $P< .10$, two-tailed test), the dextroamphetamine group showed a slower rate of learning than the placebo group. The rate of learning was not significantly different from placebo for any of the doses of magnesium pemoline. However, the higher the dosage of magnesium pemoline, the slower the mean rate of learning.

The direction of the differences between placebo and the various doses of magnesium pemoline is in agreement with what is known about the behavioral effects of magnesium pemoline in animals: higher doses inhibit learning (*9*). Only doses below this inhibitory range have been reported to facilitate behavior. In our study, however, learning rate scores under the lower doses of magnesium pemoline looked progressively more like the placebo scores. None of the results of our study indicated that in such subjects magnesium pemoline enhances learning. Other studies have indicated that acute doses of magnesuim pemoline enhance learning in rats (*3*). We did not find this to be true in human beings.

A moderately high dose of dextroamphetamine significantly slowed the rate of learning. The effects of the amphetamines on complex performance, in nonfatigued organisms, suggest that this class of stimulants interferes with performance. The amphetamines increase arousal (*10*), and high levels of arousal are detrimental to the acquisition of complex new associations (*11*). Such an interpretation may explain our findings with subjects under dextroamphetamine, since, while they learned more slowly than subjects under placebo, nevertheless the dextroamphetamine group in the simpler reaction-time task had significantly faster reaction times than the placebo group, and yet without any decrease in accuracy (*12*).

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## Reserpine and Hypothalamic Mediation

Dominic (*1*) demonstrated that the rauwolfian alkaloid reserpine, a common tranquilizer, inhibits the olfactory blockage of pregnancy induced by the urine of strange male mice. He interprets this to offer ". . . direct evidence of hypothalamic mediation in the male-induced olfactory blockage of ovoimplantation in mice." This interpretation I question; and it should be acknowledged that obtaining good evidence of this is not so facile.

Reserpine causes general reduction in responsiveness to all stimuli, including alarming stimuli, of which the urine of a strange male may be one. At a more fundamental, albeit not necessarily more relevant, level, reserpine acts physiologically by depleting stores of catecholamines and serotonin—substances thought to serve neurotransmitter or neuromodulatory functions—and by impairing mechanisms for reconstituting these stores. But this effect is by no means specific to the hypothalamus; it is manifest in all parts of the central and peripheral sympathetic nervous systems (*2*).

Hypothalamic mediation of olfactory-induced blockage of implantation is probable and had indeed been suggested before (*3*). But experimental evidence of such mediation is not provided by the effects of this relatively nonspecific drug reported by Dominic.

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Dominic [*Science* **152**, 1764 (1966)] suggests that the effect of reserpine in blocking the failure of pregnancy in newly mated female mice produced by exposure to fresh urine from alien males was due to the hypothalamic action of reserpine.

This conclusion may be sound, but in humans reserpine frequently produces marked swelling of the nasal mucosa and blockage of the nasal air-

way. Therefore I think it possible that his results could be explained by inability of the pheromones to reach the receptor sites.

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I have no evidence that reserpine in the dosage used by me caused any marked swelling of the nasal epithelim that resulted in blockage of the nasal passage. Moreover, close examination of the reserpine-dosed animals revealed no abnormality in breathing movements. Thus the possibility that the animals were rendered anosmic by administration of reserpine seems remote.

Welch raises the important question of hypothalamic mediation in the male-induced failure of pregnancy in mice and in the role of reserpine in preventing the failure of pregnancy. There is considerable evidence that, at least in the rodents, administration of reserpine inhibits the release of follicle-stimulating and luteinizing hormones and stimulates, or withdraws inhibition of, the release of prolactin from the hypophysis (*1*).

Minute amounts of reserpine, insufficient to cause hypertrophy of the mammary gland in the rabbit when administered systemically, cause secretion of milk when injected into the third ventricle (*2*). Implantation of very small amounts of solid reserpine into the posterior tuberal area of the hypothalamus in rabbits releases prolactin from the hypophysis, without noticeable damage to the brain tissue (*3*); direct implantation of reserpine into the hypophysis does not induce release of prolactin (*3*).

Reserpine does not provoke release of prolactin from the hypophysis of rabbits bearing elecrolytic lesions in the basal tuberal hypothalamus; on the contrary, reserpine induces release of prolactin if the lesions are made elsewhere in the hypothalamus (*4*). Thus it seems very likely that the release of prolactin from the hypophysis that is induced by reserpine is mediated by the basal tuberal hypothalamus.

The immediate endocrine cause of the failure of pregnancy that is induced in mice by males is the failure of the luteotrophic activity of the anterior hypophysis (*5*). Thus there is sufficient reason to believe that the inhibition by reserpine of the male-induced failure of pregnancy is caused by suppression of the inhibitory influence of the hypothalamus on pituitary release of prolactin.

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## Extraterrestrial Life

Horowitz comes close to error as well as incongruity when he dismisses (*1*) the possibility of life on Venus, limiting his comments to the search for life on Mars. I find the very different outlook of Murray and Davies (*2*) much more persuasive.

The question of the surface temperature on Venus is one on which reasonable scientists can and do differ. It is commonly and unfortunately believed that Mariner II conclusively settled the question (*3*) in favor of a high surface temperature (about 700°K); in fact it did no such thing, nor was such a claim published (*4*). Many observations of high brightness temperatures at radio wavelengths have led several atmospheric physicists to hypothesize a high surface temperature and widely varying model atmospheres (*5*); but the hypotheses remain unestablished. Other workers have suggested various nonthermal mechanisms (*6*), such as electrical-discharge phenomena, to explain the high brightness temperature. Although these mechanisms have been no better established, and although they suffer from not having been explicated with the detail (see, however, *7*) of the thermal hypotheses, they can by no means be dismissed—that is, they are not in clear disagreement with observation, the final arbiter.

Perhaps new experimental tools being exploited in this laboratory (*8*) and elsewhere (*9*) will resolve the issue, or it may be that it will remain unsettled until the U.S. (or the U.S.S.R., whose interest in Venus appears greater than ours) performs such an experiment as the parachute-borne probe of the Cytherean atmosphere proposed by NASA.

Meanwhile I commend the view on exobiology of the Space Science Board of the National Academy of Sciences which stated (*10*) that "The interpretation of the radio emission is, at least, questionable. Few planetary physicists would be surprised to hear that a nonthermal source exists."

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