Table II. *Analysis of correlation coefficients between blood pressure and pulse rate for the five centrally acting drugs*

Drugs	Correlation between cells	Correlation within cells	Correlation between subjects	Total correlation
Amphetamine	-0.702* (20)	-0.134 (231)	-0.223* (11)	-0.281* (251)
Methamphetamine	-0.565* (20)	-0.144 (231)	-0.370* (11)	-0.278* (251)
Ephedrine	-0.734* (13)	-0.136 (154)	-0.003 (11)	-0.277* (167)
Phenmetrazine	-0.147* (13)	-0.485 (154)	-0.612* (11)	-0.367* (167)
Methylphenidate	0.679* (20)	0.111 (231)	0.179* (11)	0.238* (251)

Between-cells correlation represents that attributable to the different observation periods (I) and the dose (D) as well as I × D. Within-cells correlations include between-subjects and the interactions involving subjects (S, S × I, S × D, and S × I × D). The figures in parentheses represent the degrees of freedom. The between-cells correlation coefficients were all significant when they were tested with the S × I × D interaction term, thus demonstrating conclusively that these correlations were due to drug effects and not to a relationship between pulse rate and blood pressure that could have existed in the population.[5]

*$p < 0.01$.

the slope of the regression line was not statistically significant (Fig. 2).

All drugs except ephedrine produced a significant degree of pupillary dilation; however, a valid assay was obtained only when amphetamine was compared with methamphetamine (0.92 [0.05 to 6.62]).

The effects of all drugs on pulse rate were quite complex. All drugs produced tachycardia; however, for amphetamine, methamphetamine, ephedrine, and phenmetrazine there was an over-all negative correlation between pulse rate and blood pressure (Table II). The time-action curves of amphetamine on blood pressure and pulse rate are presented in Fig. 3 and illustrate typical relationships between these variables that have also been seen with methamphetamine, ephedrine, and phenmetrazine. Both the 7.5 mg. per 70 Kg. and 15 mg. per 70 Kg. doses produced a modest increase in blood pressure and a tachycardia during the first 5 hours after administration. In contrast, the 30 mg. per 70 Kg. dose level produced a greater increase in blood pressure, and, during the time when blood pressure was maximally elevated, the mean pulse rate was not significantly different from placebo value. However, 5 hours after administration of the high dose of amphetamine, when the pressor response was decreasing, a marked tachycardia was observed. The relative bradycardia seen with the greater pressor responses was undoubtedly reflex in origin and accounts for the negative correlation between blood pressure and pulse rate. In contrast to these drugs, as can be seen from Fig. 3, all doses of methylphenidate produced a marked tachycardia, and a positive correlation was found between blood pressure and pulse rate for methylphenidate (Table II).

All drugs reduced appetite as assessed by the caloric value of food selected from the serving cart, and valid assays were obtained for all drugs. Potency estimates were not significantly different from potency estimates obtained using blood pressure for methamphetamine, phenmetrazine, and ephedrine. Methylphenidate was somewhat more potent in suppressing caloric intake than it was in elevating blood pressure but not in producing amphetamine-like subjective effects (Table I).

All drugs decreased the patients' estimates of sleep time, and valid assays were obtained for this measure for all drugs except ephedrine. The potency estimates were in good agreement with the potency estimates obtained with the physiologic measures and subjective rating scales (Table I).

Subjective effects. All drugs increased in a dose-related manner the scores on A, BG, and MBG scales (Fig. 2); however, the usefulness of these scales in calculating