crease cataplexy in patients with frequent attacks. These drugs are not specifically efficient for treatment of the auxiliary symptoms in patients. Data obtained from human studies do not negate the postulated aminergic-cholinergic interaction involved in cataplexy, and in fact nicely complement the experimental animal data.

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Does Papaverine Affect Brain Dopamine?

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Several lines of investigation have suggested that papaverine might affect brain dopamine levels. Ernst [3] reported that papaverine and bulbocapnine induced catatonia in animals and that this effect could be antagonized by levodopa and apomorphine. Based on these observations he concluded that both alkaloids are dopamine receptor blockers. More recently, Cubeddu et al [1] demonstrated that in rat neostriatal tissue cultures, papaverine induced a marked efflux of ^{3}H-labeled 3,4-dihydroxyphenylacetic acid, an effect consistent with a reserpine-like activity on dopamine storage vesicles. Further, Duvoisin [2] has observed that addition of papaverine to the treatment regimen of patients receiving levodopa for Parkinson disease resulted in gradual antagonism of the therapeutic effect of the latter.

Secretion of prolactin from the hypophysis is controlled by dopaminergic neurons in the hypothalamus [4]. Thus, agents which increase the concentration of brain dopamine, such as levodopa or the direct dopamine receptor agonist apomorphine, are known to decrease serum prolactin levels. In contrast, the dopamine antagonists such as the butyrophenones, the depleting agent reserpine, and the decarboxylase inhibitor methyldopa increase serum prolactin levels [5]. Since serum prolactin is a sensitive index of drug effects on dopaminergic neurons in the hypophysis, we investigated the effects of chronic papaverine administration on serum prolactin levels.

Twelve patients with a diagnosis of multiinfarct dementia were randomly assigned to treatment with either papaverine (Pavabid HP, Marion Laboratories), up to 1,500 mg per day, or placebo in a double-blind, parallel-groups-design study. The papaverine and placebo groups had a mean age of 64.0 and 62.2 years, respectively. Serum prolactin levels were determined by radioimmunoassay four times during the study, twice at baseline and then at the end of 30 and 60 days of treatment. For statistical analysis, the mean of the two pretreatment observations was used as the baseline.

Results of this study are presented in the Table. Analysis of variance revealed that papaverine did not significantly affect serum prolactin levels: $F(1,10) = 0.01$, $p > 0.05$. Thus, our prolactin data do not support the hypothesis that brain dopaminergic activity is influenced by administration of papaverine.

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Serum Prolactin Levels[a]

Treatment	Baseline	Day 30	Day 60
Papaverine[b]	7.66 ± 1.80	7.12 ± 1.99	7.50 ± 2.61
Placebo	7.35 ± 1.03	7.81 ± 1.09	7.82 ± 2.15

[a] Values are in nanograms per milliliter, mean ± standard error.
[b] Mean daily dose of papaverine was 1,296 mg at day 30 and 1,107 at day 60.