healthy controls. No differences were noted between the Capgras and the Frégoli groups of patients, either with respect to time taken for the accomplishment of the test or with respect to errors.

The above observations have been incorporated in the Associate Professorship Thesis of the second of us (1) and in a paper presented at the 7th Greek Congress in Neurology and Psychiatry (2), but since both communications were made in Greek, Drs Hayman and Abrams could not have been aware of them.

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RENIN AND DEPRESSION

Dear Sir,

We have read the paper by Dr Hullin and his associates, 'Renin and Aldosterone Relationship in Manic-Depressive Psychosis' (*Journal*, December 1977, **131,** 575–81). We were particularly interested to learn about the 'blunted response (of PRA) to change of posture' in patients with primary affective disorders (PAD), similar to that observed in patients with 'autonomic insufficiency and receiving β-adrenergic blocking drugs such as propranolol'.

We should like to comment that we ourselves (Altamura and Morganti, 1975) had at an earlier date reported, in patients with endogenous depression, standing PRA values significantly lower than in healthy controls, whereas recumbent PRA values were only somewhat lower than in the same healthy controls but short of statistical significance. In those patients, treatment with lithium salts tended to raise both recumbent and standing PRAs. In that paper, also, we put forward the hypothesis of a reduced function of β-adrenergic receptors in endogenous depression. More recent data, comparing PRA values in patients with primary and secondary depression (Altamura *et al*, 1977) apparently confirm our own earlier observation as well as those of Dr Hullin *et al*, namely that orthostatic stimulation would produce poor activation of the renin-angiotensin system in patients with primary depression, as it does in patients treated with β-adrenergic blocking agents—the same agents, in turn, producing depressive states (Waal, 1967). There is, however, a discrepancy between our recent findings and Dr Hullin's, i.e. that our patients with endogenous depression showed no tendency to increased recumbent PRA values but indeed the opposite. This may be explained in two ways. One is that Dr Hullin *et al* drew their data from a group of only three patients, probably not enough to warrant final conclusions. And the other is that all three of Dr Hullin's patients might have been bipolars with rapid mood switches. Last, we may add that our follow-up observations of PRA values in three patients receiving long-term lithium therapy indicate continuing high values for both standing and recumbent positions after more than two years of treatment.

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