suggests that both adrenaline and noradrenaline may be displaced by their α-methyl homologues. The fact that α-methylnormetanephrine does not appear in the urine for 10 to 12 hr. is consistent with the α-methylnoradrenaline's being held at nerve endings until the noradrenaline is displaced.

A further point of interest was that there was a striking increase in the excretion of vanillic acid during these experiments. Eight hr. after the α-methyldopa was given, the vanillic acid excretion was about ten times greater than in the control period. The acid could not have arisen from exogenous sources since the patients were fasted for the early part of the test and the food taken later was known to be free from sources of vanillic acid.

This observation suggests that vanillic acid can arise from α-methylnoradrenaline by an analogous series of reactions to that in which it is known to arise from noradrenaline (Smith and Bennett, 1958; Rosen, and Goodall, 1962).

The results of these studies will be published *in extenso* at a later date.

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Effects of Reserpine in Rats Pretreated with α-Methyldopa

SIR,—Day and Rand (1963a) have described an arousing effect of α-methyldopa in rats sedated by reserpine. It was assumed that, when the brain stores of dopamine and noradrenaline had been emptied by reserpine, there was a repletion by the α-methylated catecholamines formed from α-methyldopa. Carlsson and Lindqvist (1962) have shown that α-methyldopa is decarboxylated *in vivo* and that α-methyldopamine is transformed into α-methylnoradrenaline in the brain. It was suggested by Day and Rand (1963b) that the α-methylated catecholamines may serve as "false transmitters", probably with a less potent activity than the natural amines.

In the present study we have further demonstrated a central action of the α-methylated catecholamines by pretreating rats with α-methyldopa before a depletion of the catecholamine stores is produced by reserpine.

The rats, weighing 300 g. were given α-methyldopa (Aldomet, Merck, Sharp and Dohme Ltd.) 150 mg./kg. intraperitoneally at 9 a.m. on the first and second days, followed by 300 mg./kg. at 4 p.m. on the second day. On the third day these pretreated rats, together with untreated control rats, were given reserpine, 2 mg./kg. i.p. Before the reserpine injection all animals appeared normal.

The controls were deeply sedated after reserpine, with decrease in muscular tone, eyelid ptosis and diarrhoea. Pretreatment with α-methyldopa before reserpine administration, however, resulted in the occurrence of exophthalmos (for 4–5 hr.), an increase in muscular tone with a state resembling catatonia (for 3–4 hr.) and an absence of diarrhoea (for 8–24 hr.). After 24 hr. all rats were mildly sedated and had diarrhoea. At this time the animals pretreated with α-methyldopa could no longer be distinguished from the controls given reserpine only.

The administration of a potent catechol-*O*-methyltransferase inhibitor α-propyldopacetamide (H22/54 Hässle Ltd.) 300 mg./kg. immediately before and 4 hr. after the administration of reserpine, did not influence the appearance or the duration of the symptoms. In a second series of experiments rats were pretreated with α-methyl-*m*-tyrosine in place of an equal dose of α-methyldopa. The results obtained from rats administered α-methyl-*m*-tyrosine plus reserpine, however, did not differ from those of the reserpine treated controls.

The syndrome noticed after reserpine administration in rats pretreated with α-methyldopa has been tentatively ascribed to a liberation of α-methylated catecholamines in the brain. Since α-methyldopamine is known to disappear from the brain within 16–20 hr. after the administration of α-methyldopa, the signs noted are probably caused by α-methylnoradrenaline. This substance is not attacked by monoamine oxidase, a fact which may account for the prolonged action observed in this experiment. When reserpine depletes the brain stores of naturally occurring catecholamines there are only rapidly transient signs of sympathetic activity. The absence of corresponding signs and symptoms after pretreatment with α-methyl-*m*-tyrosine may indicate that its corresponding decarboxylation product (metaraminol) is devoid of central action.

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Banana and Restraint Ulcers in Albino Rats

SIR,—Banana has been reported to markedly decrease histamine induced gastric acidity (Sanyal, Das, Sinha and Sinha, 1961), and to have prophylactic value in phenylbutazone-induced gastric ulcers in guinea-pigs (Sanyal, Gupta and Chowdhury, 1963). Encouraged by these results it was thought worthwhile to screen the efficacy of banana in another test preparation which simulates human peptic ulcer in its pathogenesis. Restraint ulcer technique (Brodie and Hanson, 1960) was selected, because the lesions produced by this method are consistently in the glandular portion of the stomach and are the result of a physiologically induced stress.

Male albino rats (140 to 170 g.) were divided into two groups. One group of 10 rats acted as control. In the second group of 15 rats the usual diet was