of a distribution are met most satisfactorily when the observations of the noradrenaline content of the spleen are considered without transformation.

It is possible that differences in the regional distribution of noradrenaline in the cat spleen underlie the difference between our results with undivided spleens and those of Brown & others (1967) relating to divided spleens. Certainly, differences in the densities of the adrenergic innervation of the lateral (thin or anterior) and medial (wide or posterior) ends of the cat spleen have been suggested (Green & Fleming, 1968).

This work forms part of a thesis presented by one of us (M.I.R.) in fulfilment of the requirements for the Degree of Doctor of Philosophy in the University of London.

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Effect of calcium on reserpine-induced catalepsy

High calcium pretreatment reverses the gross behavioral effects of reserpine in the guinea-pig as well as the reserpine-induced inhibition of pethidine analgesia (Radouco-Thomas, 1971). Furthermore, calcium pretreatment attenuates reserpine rigidity (Radouco-Thomas, 1970) and partially antagonizes the reserpine-induced depression of the conditioned avoidance response in the rat (Boyaner & Radouco-Thomas, 1971). These results prompted an investigation of the effect of high calcium pretreatment on reserpine-induced catalepsy in the rat.

Sprague-Dawley, male rats (260-300 g) were used to assess the intensity of catalepsy by carefully raising each leg of the animals in turn to a height of either 2 or 5 cm by placing it on a suitable block. If the rat did not remove its leg within 15 s, the catalepsy test was taken to be positive. The degree of catalepsy was expressed as the percentage of positive responses obtained in each group.

The rats were randomly divided into four treatment groups. Each group consisted of a minimum of 9 animals: group 1—placebo (0·9% NaCl); group 2—calcium chloride (3 × 100 mg/kg calcium); group 3—reserpine (1 mg/kg); group 4—calcium plus reserpine (3 × 100 mg/kg calcium +1 mg/kg reserpine).

All drugs were administered subcutaneously. The three injections of calcium were given at 15 min intervals. In calcium-reserpine treated rats, reserpine was injected 15 min after the last calcium injection.

Calcium-treated rats showed only a few positive responses (10% at 6 h) in the 2 cm block test and none in the 5 cm block test. The reserpine-treated group demonstrated the maximum intensity of catalepsy for the percentage of positive responses on both tests exceeded 50% at 3 h (56 and 59% respectively) and reached 100% at 6 h. On the other hand, in the calcium-reserpine treated group, the percentages of positive responses in the two tests were 36 and 19% at 3 h, and 44 and 53% at 6 h, respectively. Thus, calcium pretreatment resulted in a slower progression and an attenuation of the reserpine-induced catalepsy.

A dopamine deficiency in the extrapyramidal centres has been implicated in reserpine-induced catalepsy in animals and in man, for L-dopa readily reverses this syndrome (Carlsson, Lindqvist & Magnusson, 1957; Hornykiewicz, 1966; Blaschko & Chruschiel, 1960; Degkwitz, Frowein & others, 1960; Larochelle, Bedard & others, 1971). This attenuation of reserpine-induced catalepsy by exogenous calcium could be due to its inhibition of reserpine-induced depletion of cerebral dopamine as well as noradrenaline and 5-HT (Radouco-Thomas, Tessier & Lajeunesse, 1971).

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