monophosphate (cAMP) content of the superior cervical ganglion (McAfee, Schorderet & Greengard, 1971). To determine whether this rise in cAMP could play a part in the control of transmitter synthesis we have investigated the effect of $N^6,O^{2'}$-dibutyryl adenosine 3′:5′-cyclic monophosphate (dbcAMP), a stable analogue of cAMP, on the synthesis of NA in rat superior cervical ganglia *in vitro*.

Superior cervical ganglia were removed from albino Wistar rats (250 g) under urethane anaesthesia. The ganglia were desheathed on ice and then incubated at 37° under O_2/CO_2 in 1 ml of Krebs bicarbonate containing amino acids, vitamins, 3% calf serum and, where appropriate, 1 mM dbcAMP.

Ganglia isolated in this way contained $17{\cdot}8 \pm 1{\cdot}3$ ng NA. After 2 h incubation the NA content of control ganglia was $23{\cdot}0 \pm 1{\cdot}8$ ng ($n=8$) and of dbcAMP-treated ganglia $42{\cdot}9 \pm 4{\cdot}1$ ng ($n=8$), an increase of 87% over the control ($P<0{\cdot}01$). These levels were maintained for at least 8 h. Theophylline (1 mM) and cAMP (5 mM) also caused a rise in NA content. Cycloheximide (20 μg/ml) did not block the effect of dbcAMP on NA levels showing that this effect did not require the synthesis of new protein.

If, 2 h after the start of incubation when NA levels had reached a new steady state, NA synthesis was stopped by addition of α-propyldopacetamide (10^{-4}M), NA levels in control and dbcAMP-treated ganglia declined at the same rate ($t_{1/2}$ approximately 4 h) showing that dbcAMP did not produce its effect by slowing the release or breakdown of NA. This suggested that dbcAMP might be acting by increasing NA synthesis. To test this ^{14}C-L-tyrosine was added to the incubation medium 2 h after the commencement of incubation which was continued for a further 1 h. The ganglia were homogenized and the ^{14}C-dopa, -dopamine and -NA formed were separated by adsorption to alumina followed by differential elution from Amberlite CG-120 resin. In dbcAMP-treated ganglia the ^{14}C-NA synthesized was $5{\cdot}75 \pm 0{\cdot}62$ (nmoles/g wet wt)/h ($n=11$) as compared with $3{\cdot}11 \pm 0{\cdot}39$ (nmoles/g)/h in control ganglia ($n=12$) an increase of 85% ($P<0{\cdot}01$).

These results suggest that cAMP may play a role in the short-term control of NA synthesis in the cell body of the sympathetic neurone.

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The influence of growth and of adrenalectomy upon some rat heart enzymes

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Monoamine oxidase (MAO) activity in the rat heart increases with the age of the animal (Horita, 1968), and also, together with $NADH_2$ cytochrome C reductase (NCR), following adrenalectomy (Callingham & Della Corte, 1971). In this study the effects of these factors on the half-lives of both enzymes have been examined.

Male Wistar rats were used. They were adrenalectomized under ether anaesthesia and maintained on 0·9% sodium chloride solution. MAO assays were per-

formed radiochemically, using ^{3}H-tyramine as substrate. NCR was estimated both fluorometrically and in the spectrophotometer.

Both MAO and NCR were found to increase with the age of the animal which in the age groups studied, closely paralleled the increase in the weights of the hearts. Following adrenalectomy there was a further increase in enzyme activity superimposed upon that due to the growth of the animal. This increase reached a maximum 14 days after operation. The MAO remained at this level for at least a further 14 days, but the NCR activity began to fall towards normal within a week.

The half-life of the MAO in the hearts of both adrenalectomized and normal animals was measured by irreversibly inhibiting the enzyme with pargyline (25 mg/kg, S.C.) and following the synthesis of new enzyme over a period of several days. Three groups of adrenalectomized rats were used together with matched controls. The first group, with a mean body weight of 155 g, were given pargyline immediately after operation. The half-life of the MAO was 6·2 days compared with 6·7 for the controls. The second group were pargyline treated 13 days after adrenalectomy. The MAO half-life was now 10·1 in the operated animals compared with 10·2 for the controls, for a mean body weight of 230 g. In the third group, with a mean body weight of 340 g, the MAO half-lives were 15·4 and 17·6 days in adrenalectomized and control animals respectively. At no time was there a significant difference in the half-life of the enzyme between operated and control rats.

Calculations of the half-lives of both MAO and NCR from the changes in enzyme activity following adrenalectomy gave values of 6·3 and 6·5 days respectively for the two enzymes in animals weighing 155 g. The breakdown of NCR calculated from its return to the normal level gave a half-time of 11·5 for rats weighing 260 g. All values obtained for the half-lives of both enzymes showed a close linear relationship to either the body weight or to the heart weight of the animals.

As the rats grow older, the rate of destruction of both MAO and NCR decreases, which may account for the observed increase in enzymic activity seen with age. Adrenalectomy has no apparent effect upon this ageing process.

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Catecholamine induced contractures in denervated muscle

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Chronically denervated skeletal muscle has been shown to produce contractures in response to applied catecholamines both *in vivo* (Bowman & Raper, 1965;