and water. Systolic blood pressure was measured in the unanaesthetised rat by the tail cuff method (Bunag, 1973) between 08.00 and 10.00 h on one day and again 48 h later; the rats were killed by cervical dislocation 24 h after the second blood pressure measurement. Blood was collected immediately by heart puncture (in the youngest group blood was collected by decapitation). Renin concentration in plasma was measured by radioimmunoassay for generated angiotensin I using a modification of the method of Poulsen & Jorgensen (1974). The results are summarised in Table 1 and indicate that the levels of renin in the plasma of NR and SHR initially decrease with increasing age until the rats are 44 to 46 days old, but rise subsequently. Following the initial decrease, the rate of rise in plasma renin levels in the SHR was more pronounced than that in the NR, and the levels of renin of the SHR after 90 days were about twice those of the NR. It is concluded that plasma renin levels change with increasing age in NR and SHR and that the changes follow a similar pattern.

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Effect of centrally administered noradrenaline and isoprenaline on splanchnic nerve activity in anaesthetised and conscious cats

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The recording of splanchnic nerve activity in various laboratory species has been used as an index of sympathetic tone in order to demonstrate a central action of some antihypertensive drugs. For instance, Schmitt, Schmitt & Fénard (1974) reported a reduction in splanchnic nerve activity in the conscious dog following i.v. injection of clonidine, and Lewis & Haeusler (1975) observed a fall in splanchnic nerve activity after i.v. infusion of propranolol in the conscious rabbit. We have recorded splanchnic nerve activity in both consious and anaesthetised cats in order to provide further evidence to support the contention (Day, Poyser & Sempik, 1976) that pressor responses evoked by the administration of noradrenaline or isoprenaline into the third cerebral ventricle were elicited by actions of these substances on structures within the brain.

The experiments were performed in 6 anaesthetised (chloralose 70 mg/kg i.v.) and 6 conscious cats. Blood pressure was recorded from a femoral artery in anaesthetised animals and from an indwelling carotid catheter in conscious animals; heart rate was obtained from the blood pressure pulse. Nerve activity was recorded from bipolar tungsten electrodes implanted on the greater splanchnic nerve by a method similar to that described by Haeusler & Lewis (1976). In both series of experiments cannulae were inserted stereotaxically into the third ventricle.

In the control situation bursts of electrical activity recorded from the splanchnic nerve coincided with respiration and the amplitude of the discharges varied between 20–120 μV in each experiment.

I.v. injection of noradrenaline (0.5 – 1 μg/kg) caused a brief rise in arterial blood pressure associated with a reflex bradycardia and a fall in splanchnic nerve activity. On the other hand, infusion of noradrenaline (15 μg) into the IIIrd ventricle of anaesthetised cats caused an increase in blood pressure and heart rate with a concomitant increase of 30–40% in splanchnic nerve activity. Noradrenaline (5–10 μg) infused into the IIIrd ventricle of conscious cats produced similar effects on the cardiovascular system and on sympathetic nerve activity.

Infusion of isoprenaline (5 μg) into the IIIrd ventricle of conscious cats caused rises in arterial blood pressure and splanchnic nerve activity; the time course of these responses was similar to those produced by equipressor amounts of noradrenaline administered by this route.

These results provide further evidence of the central nature of the pressor responses to III ventricle noradrenaline and isoprenaline in the cat.

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The effect of atenolol on the discharge of sympathetic efferent nerves in the anaesthetised cat

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The mechanism of action of β-adrenoceptor blocking drugs in hypertension remains obscure. A central action was suggested by Lewis & Haeusler (1975) who demonstrated that propranolol reduced sympathetic efferent discharge. The effect of the β-adrenoceptor antagonist atenolol, which has a low lipid solubility and therefore is not thought to cross the blood brain barrier (Barrett, 1977) on sympathetic efferent discharge (SED) was investigated.

Cats were anaesthetised with α-chloralose (80 mg/kg i.p.) and artificially ventilated. Temperature and the pH, pCO_2 and pO_2 of the arterial blood were maintained within normal limits. Recordings were made of the discharge from few-fibre preparations dissected either from the lumbar trunk or the renal nerves. The mean arterial blood pressure in six cats during the control period was 105 ± 6 mmHg (mean ± s.e. mean) and the SED was 12.4 ± 4.4 impulses/s. Blood pressure was raised or lowered by the administration of phenylephrine (1–4 μg/kg) or glyceryltrinitrate (2–20 μg/kg) and the SED was recorded under steady state conditions over a range of widely differing blood pressures. Atenolol (3 mg/kg i.v.) was administered when the blood pressure had returned to initial levels. Thirty minutes later the mean blood pressure was reduced to 98 ± 7 mmHg ($P < 0.01$) and in spite of this the SED was reduced to 4.9 ± 1.5 impulses/s ($P < 0.05$). The blood pressure was again artificially raised and lowered and the SED recorded over the same range of blood pressures as before. Changes in SED are shown in Figure 1. The responses of fibres from the renal nerves and the lumbar trunk were similar.

It is concluded that atenolol reduces the SED. A similar conclusion has recently been reached by Friggi, Chevalier-Cholat & Bodard (1977). In addition atenolol may also attenuate the reflex response to fluctuations in blood pressure and this may play a role in its antihypertensive action.

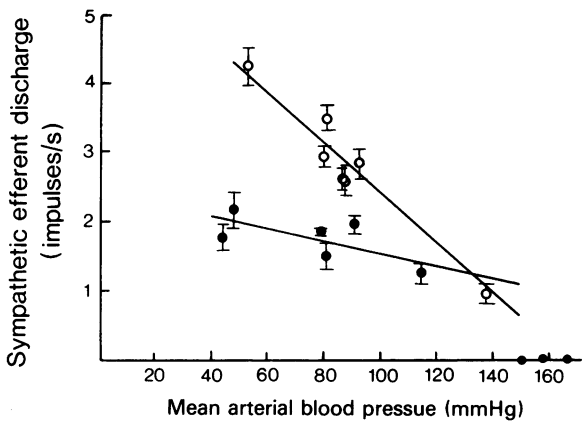

Figure 1 Effect of atenolol on SED recorded from the lumbar trunk of a cat over a range of widely differing blood pressures. (○) show measurements made before the administration of atenolol (3 mg/kg) and (●) at least thirty minutes after administration of atenolol. Each point shows the mean of between 6 and 14 measurements and the vertical line represents s.e. mean.

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