mmHg. The hepatic portal venous flow (HPVF) was 241.7 ± 63.2 ml/min and the hepatic portal venous pressure (HPVP) 5.42 ± 1.82 mmHg. The hepatic portal vascular resistance (HPVR), calculated as (HPVP–IVCP)/HPVF was 0.017 ± 0.008 mmHg ml^{-1} min, or 0.051 ± 0.026 mmHg ml^{-1} min 100 g liver weight.

Isoprenaline was injected on 39 occasions in 9 preparations (100 ng–100 μg intraportally): in no experiment was there a dose-dependent reduction in HPVR, the maximum fall being under 5%, and doses over 1 μg tended to cause small increases in HPVR. Similar responses were obtained when a background portal 'tone' was induced by intraportal infusions of noradrenaline (1, 5 & 10 μg/min).

Phenylephrine, noradrenaline and adrenaline were injected intraportally in graded increasing doses: the only response was portal vasoconstriction. The thresholds, and doses which doubled the HPVR were: phenylephrine ($n = 4$) 0.5 to 5.0 μg and 6.3 (± 3.0) × 10^{-7} mol (mean ± s.e. mean); noradrenaline ($n = 4$), 100 ng and 2.5 (± 0.8) × 10^{-8} mol; adrenaline, the most potent, ($n = 4$), 100 ng and 6.5 (± 0.3) × 10^{-9} mol.

The form and position of these dose-response curves to intraportal phenylephrine, noradrenaline and adrenaline were unaffected by propranolol (250 μg/kg, i.v.): the doses of each substance required to double the HPVR were not significantly altered by β-adrenoceptor blockade ($P>0.30$, $n = 3$, paired t-test). In contrast, phentolamine (1.0 mg/kg. i.v.) caused a shift of the portal vasoconstrictor dose-response curves for all three substances to the right: the mean dose-ratios for the doubling in HPVR before and after the α-adrenoceptor antagonist were 32.4 for phenylephrine, 12.1 for noradrenaline and 20.1 for adrenaline, and were not significantly different for any pair of the three substances ($P>0.20$; $n = 4$).

The canine portal venous vascular bed contains α-adrenoceptors which, when stimulated, cause portal vasoconstriction, but β-adrenoceptors are not present in this bed in an adequate population either to mediate a portal vascular response to intraportal injections of isoprenaline, or to modulate the portal vasoconstrictor responses to adrenaline or noradrenaline. This adrenoceptor population contrasts with that of the hepatic arterial bed which possesses both α and β-adrenoceptors (Richardson & Withrington, 1977b).

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Receptors mediating hyperpolarizing responses to catecholamines in rat superior cervical ganglia

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The receptors mediating the hyperpolarizing action of catecholamines on sympathetic ganglion cells have not yet been fully characterized. The present communication describes some attempts to define the receptors responsible for the hyperpolarization of rat sympathetic ganglion cells produced by adrenoceptor stimulants *in vitro*. Potential changes in superfused desheathed rat superior cervical ganglia were recorded extracellularly by the 'air-gap' method of Brown & Marsh (1974), with temperature controlled to 25°C ± 1°C.

The sympathomimetic amines listed in Table 1 produced a rapid, reversible low-amplitude (≥400 μV) hyperpolarization. Clonidine (10^{-9} to 10^{-6} M) and ergometrine (10^{-8} 10^{-5} M) also produced a hyperpolarization which was much more persistent (2–3 h) and had characteristics of partial agonism.

Table 1 Hyperpolarization produced in rat superior cervical ganglia by sympathomimetic amines (a) EC_{50} values (b) antagonism by phentolamine

	a *Agonist* EC_{50} (μM)	*b* *Phentolamine* (10^{-6} M) *dose ratio*
(−)noradrenaline	$1.7 \times 10^{-6} \pm 0.6$ (5)	5.6 ± 0.9 (4)
(±)isoprenaline	$4.1 \times 10^{-6} \pm 0.8$ (6)	157.3 ± 48.1 (4)
(−)phenylephrine	$4.2 \times 10^{-6} \pm 0.4$ (4)	24.7 ± 3.8 (5)
ADTN*	$6.7 \times 10^{-6} \pm 0.4$ (3)	1.3 ± 0.3 (3)
dopamine	$1.7 \times 10^{-5} \pm 0.5$ (4)	2.3 ± 0.1 (4)
(±)amidephrine	$1.1 \times 10^{-3} \pm 3.1$ (3)	

Figures in brackets represent number of experiments.
Values are given as means ± s.e.means.
*2-amino, 6,7-dihydroxytetralin.

Phentolamine (10^{-6} M) produced a variable degree of antagonism, as measured by the dose-ratio (Table 1*b*). Isoprenaline (most sensitive to phentolamine) was resistant to propranolol (10^{-6} M), suggesting the absence of β-receptors. Variations in the effect of phentolamine might reflect (*a*) the presence of dopamine receptors (selectively stimulated by dopamine and ADTN) or (*b*) influence of uptake processes (cf. Langer & Trendelenburg, 1969). The α-receptors might resemble 'presynaptic' α-receptors, in view of the action of clonidine.

M.P.C. is an M.R.C. Student.

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Do cyclic nucleotides mediate slow postsynaptic potentials in sympathetic ganglia?

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In sympathetic ganglia, it has been proposed that the slow EPSP (excitatory postsynaptic potential) is mediated by cyclic GMP (guanosine 3′,5′-monophosphate) and the slow IPSP (inhibitory postsynaptic potential) by cyclic AMP (adenosine 3′,5′-monophosphate (McAfee & Greengard, 1972). To test this hypothesis we used the sympathetic ganglion of the bullfrog, where the slow PSPs occur in different cell types; the slow EPSP in type B cells and the slow IPSP in type C cells. We applied cyclic GMP and cyclic AMP to determine whether they mimicked the membrane potential and conductance changes involved in the slow postsynaptic potentials. We also used the monobutyryl, dibutyryl, 8-bromo and 8-parachlorophenylthio derivatives of these cyclic nucleotides, which have greater membrane permeability and/or resistance to inactivation by phosphodiesterase. Using the sucrose gap recording technique, no consistent responses to cyclic GMP or its derivatives (1 mM) were observed in 80 tests on 20 preparations. In only 3 preparations was a depolarizing response observed. Additionally, no consistent responses to cyclic AMP or its derivatives (1 mM) were observed in 14 tests on 8 preparations. With intracellular recording from individual B or C cells, responses to the administration of the cyclic nucleotides or their derivatives were also rare, although responses to acetylcholine were observed. The cyclic nucleotides were administered by perfusion, as well as by extracellular or intracellular iontophoresis. In the few cells where responses were observed, there was little correlation between the potential and/or conductance change of the PSP and the response to the cyclic nucleotide. For example, of the 52 B cells tested, 46 had no consistent responses to cyclic GMP or its derivatives. Of the remaining 6 cells,