latency than saline controls (trial 1, saline mean latency 12 ± 1 s, ethanol 7 ± 1 s; trial 2, saline 137 ± 11 s, ethanol 23 ± 7 s; $P < 0.001$). Dose response relations could be demonstrated in both trials for doses of ethanol between 0.9 and 2.0 g/kg ($n = 15$ or 20 per group). If rats were not shocked in the dark compartment at trial 1, latencies at trial 2 did not differ significantly from trial 1. Pre-treatment with α-methyl-*p*-tyrosine (80 mg/kg, i.p.) 3.5 h before ethanol or saline did not affect the ethanol induced changes at either trial ($n = 17$ per group).

The test procedures in both kinds of experiment described depended on the initiation of movement. In the active avoidance task ethanol led to quicker learning, and this effect was abolished by pre-treatment with α-methyl-*p*-tyrosine, which suggests that a newly synthethized catecholamine may have been involved. In the passive avoidance task ethanol hindered learning and this was not affected by α-methyl-*p*-tyrosine.

Although the effect of ethanol on the performance of both these tasks in rats can be described as 'stimulating' movement, each is presumably mediated by different neuronal pathways.

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Effect of iontrophoretic and intravenously administered atropine on the acetylcholine-discharges of lateral geniculate neurones

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In the present study, acetylcholine (ACh) sensitivity of lateral geniculate cells was re-examined after iontophoretically and intravenously administered atropine under different experimental conditions.

Conventional techniques for extracellular recording with 5-barrel micropipettes and iontophoretic drug application were used (Krnjević & Phillis, 1963). The outer barrels were filled with the following substances: ACh (1 M, pH 4.9), atropine sulphate (10 mM in 165 nM NaCl, pH 5.5), mecamylamine HCl (10 mM in 165 mM NaCl, pH 6.6) and L-glutamate (1 M, pH 7–7.4). Geniculate neurones of lamina A were identified by the visual evoked response and by the neuronal sensitivity to L-glutamate and ACh. Subsequently, dye deposition was achieved to mark recording sites and to permit the histological reconstruction of electrode tracks (Godfraind, 1969, 1976; Godfraind & Meulders, 1969). All animals, except one, received an injection of atropine methylnitrate (4 mg/kg, i.v.), and were paralyzed with an intravenous infusion of succinylcholine at an approximate rate of 1 mg/min.

In an anaesthetized cat (fluothane 1%), and in a non-anaesthetized midpontine pretrigeminal preparation, the observations made were in agreement with previous descriptions: neuronal discharges induced by ACh were depressed either by atropine or by mecamylamine applied by iontophoresis, as well as after an intravenous injection of atropine sulphate (3 mg/kg) (Curtis, 1966; Curtis & Crawford, 1969; Marshall & McLennan, 1972).

Different results were observed under α-chloralose (80 mg/kg, i.v.; 3 cats) and urethane (1 g/kg, i.v.; 2 cats) anaesthesia. Under these conditions, discharges evoked by ACh (15–45 nA, 10–20 s) were also prevented or greatly reduced by iontophoresis of either atropine (10–20 nA, 40–50 s) or mecamylamine (10–30 nA, 30–65 s). However, after atropine sulfate i.v. administration (3 mg/kg), neuronal discharges could still be induced by ACh applied with similar iontophoretic parameters. Tests with cholinergic antagonists applied by iontophoresis were again performed on the same units to analyse the pharmacology of these 'remaining' ACh responses. These appeared to be more resistant to iontophoretically applied atropine than before the i.v. atropine sulphate injection. Indeed, after atropine had been iontophoretically reapplied with the same parameters, some of the ACh responses were comparable to the control, while others were slightly delayed by about 3 to 5 s. On the contrary, mecamylamine was a good antagonist: 7 to 30 nA mecamylamine for about 30–65 s was sufficient to depress ACh induced activity (20–45 nA ACh for 10–20 s). This antagonism was shown to be reversible provided a small dose of mecamylamine was applied.

In conclusion, the present results indicate that α-chloralose or urethane anaesthesia combined with the intravenous administration of atropine sulphate, provide favourable experimental conditions for the study, in the thalamus, of ACh responses which might be attributed to the stimulation of nicotinic receptors.

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The effect of cholinomimetic drugs on the isolated hemisected immature rat spinal cord

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Motoneuronal responses of the isolated hemisected immature rat spinal cord (Otsuka & Konishi, 1974) were measured as changes in ventral root polarity. Drugs were dissolved in 2 ml Ringer solution and perfused on to the isolated hemicord at 0.8 ml/minute. The temperature of the preparation was maintained at $20 \pm 0.5°C$.

Acetylcholine (1–10 mM) evoked depolarizing responses whereas choline (1–10 mM) had no effect. Continuous perfusion with neostigmine (1 μM) produced a small depolarization which levelled off and at this stage depolarizing responses to 10–100 μM acetylcholine could be obtained.

It was surprising to find that responses to acetylcholine were still elicited from hemicords treated with 1 mM procaine or 0.1 μM tetrodotoxin; an indication that motoneurones were responding to cholinomimetics directly.

Responses to 50 μM acetylcholine were abolished by atropine (1 μM) and were unaffected by tubocurarine (10 μM) or hexamethonium (10 μM). Thus is would appear that motoneurones of the immature rat possess muscarinic receptors. Atropine sensitive acetylcholine responses of cat motoneurones *in vivo* have been demonstrated recently by Zieglgänsberger and Bayerl (1976).

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