1971) classifying the acts and postures described by Grant & Mackintosh (1963) as non-social (exploration, grooming, etc.), social approach (including aggression), and escape from the other rat.

Nitroglycerin was administered as a 10% solution in corn-oil, syringing 0.5 ml/kg on to the shaved flank (Clark & Litchfield, 1967) 30 min after removing water and 15–30 min before test. Treating only one rat of each pair, nitroglycerin did not affect social behaviour. When both were treated, the total number of actions of all kinds observed in 3 min averaged 262 (± 21 s.e. mean) for the six pairs of nitroglycerin-treated rats. This was a decline of 20 ± 20 actions from the pre-experimental baseline, while corn-oil controls increased by 48 ± 26 to 302 ± 24 . The difference was as predicted (t=2.04, P<0.05 1-tailed) but was mainly in social approach, with little change in non-social or escape behaviour.

Conditioned aversion: Instead of water, rats were offered a solution of 0.1% saccharin sodium on the day nitroglycerin was first administered. Comparing water on the previous day with saccharine on day 2 (21 h after dosing), controls increased intake by 10.2 ± 1.4 g, rats given nitroglycerin by only 7.1 ± 1.6 g (t=1.81, df=20, P<0.05 1-tailed).

Tolerance has been reported to nitroglycerin with supersensitivity following withdrawal, and another conditioned aversion experiment tested this.

On days 1-5 rats were offered 0.1% saccharin for 1 h in the morning, treated with nitroglycerin (50 mg/kg) or ethylene glycol dinitrate (EGDN) or

corn-oil (n=10) and later offered water for 1 hour. Groups differed little in intake of either fluid, and were left untreated on days 6 and 7. On day 8 they were offered saccharine given a novel flavour with coffee and dosed again. On day 9, corn-oil alone increased coffee intake by 6.1 ± 2.5 g, EGDN by 0.1 ± 1.5 g and nitroglycerin reduced it by 0.9 ± 2.1 g ($F_{2,27}=3.39$, P<0.05).

Effects in both social behaviour and conditioned aversion tests were in the predicted direction but were much smaller than expected, perhaps because the high solubility of nitroglycerin in corn-oil reduced the effective dose.

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The release of endogenous amino acids from the cat spinal cord in vivo

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Perfusion of the central canal of the cat spinal cord in vivo is a useful approach to the study of spinal neurotransmission (Morton, Stagg & Webster, 1976). Employing this technique, Jordan & Webster (1971) demonstrated the release of acetylcholine and radiolabelled glycine into the perfusate (in the presence of eserine and p-hydroxymercuribenzoate, respectively) during stimulation of femoral and sciatic nerves.

In the present experiments, cats were spinalized at C1 under halothane anaesthesia, immobilized with gallamine triethiodide and the central canal (L4-S1) was perfused (0.06 ml/min) with artificial CSF

(Feldberg & Fleischhauer, 1960). A bipolar platinum electrode (tip separation 5 mm) was inserted into the cord at C2 to deliver trains (0.1 Hz, 5 s duration) of stimuli (12V, 2 ms, 40 Hz) for 10 min periods. Samples of perfusate were collected (10 min periods) and assayed for amino acids by a modification of the dansylation procedure described by Briel & Neuhoff (1972), utilizing [3H]-dansyl chloride with [14C]-leucine as an internal standard.

Table 1 shows those amino acids studied. Introduction of p-chloromercuriphenylsulphonate (pCMS) 10⁻⁴ M into the artificial CSF, in an attempt to inhibit amino acid uptake (Balcar & Johnston, 1973), significantly increased the efflux of several amino acids and further significant increases resulted from stimulation of descending spinal tracts. Stimulation in the absence of pCMS did not consistently increase the efflux of any amino acid studied (values not shown). The stimulated increases in glutamate, glycine, GABA and alanine efflux could be related to their proposed neurotransmitter roles (Curtis & Johnston, 1974) although the evoked release

Table 1 Release of endogenous amino acids from the cat spinal cord in vivo

Amino acid	Release following:	
	pCMS 10 ⁻⁴ м (% spontaneous)	pCMS 10 ⁻⁴ m + stimulation (% pCMS 10 ⁻⁴ m release)
	A	В
Leucine	186 ± 26 (10)†	137 ± 11 (4)‡
Alanine	$233 \pm 57(7)$	229 ± 67 (4) ‡
Aspartate	179 ± 30 (8)*	$264 \pm 102 (3)$
GABA	387 ± 98 (10)*	163 ± 11 (4)§
Glutamate	197 ± 25 (8)†	186 ± 13 (3) ‡
Glutamine	100 + 6 (10)	Not calculated
Glycine	850 ± 142 (10)†	194 + 41 (4) ±
Lysine	72 + 8 (5)*	130 ± 21 (3)
Proline	256 + 60 (6)*	169 + 29 (4)

Spontaneous release of amino acids was determined in the absence of pCMS. The ³H radioactivity in each dansyl derivative was corrected for recovery by means of a [¹⁴C] leucine internal standard and values were normalized with respect to the spontaneous efflux of endogenous leucine.

- A: Release after 60 min perfusion with pCMS 10⁻⁴ m in artificial CSF. Values are per cent of spontaneous release.
- B: Maximum release following stimulation in the presence of pCMS 10⁻⁴ M. Values are per cent of pCMS 10⁻⁴ M release.

All values are mean \pm s.e. mean (number of observations in parentheses). Significance levels (2-tail paired t-test): $P < 0.05^*$ or 0.01 \ddagger compared with spontaneous release. $P < 0.05\ddagger$ or 0.01 \ddagger compared with release following pCMS 10⁻⁴ M. For all other comparisons P > 0.1.

of leucine (not a neurotransmitter candidate) indicates that caution is necessary in this interpretation.

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The effect of cortisol on α_1 -macroglobulin and α_2 -acute phase globulin in the arthritic rat

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Recent work has shown that plasma antiproteases may be anti-inflammatory since they inhibit a number

of proteases that are involved in inflammation (Barrett & Starkey, 1973; Hercz, 1974). The two most abundant human plasma antiproteases are α_1 -antitrypsin and α_2 -macroglobulin. In rat plasma the corresponding pair of antiproteases are α_1 -antitrypsin and α_1 -macroglobulin. A third antiprotease, α_2 -acute phase globulin, is produced as a result of inflammation. Both α_1 -macroglobulin and α_2 -acute phase globulin are similar in properties to human α_2 -macroglobulin and can be assayed enzymatically by