Exposure to ethanol (10% v/v for 10 to 20 min) 'anaesthetized' the leeches (they became unresponsive to noxious stimuli) without altering the ACh content of the nerve cord (Table 1). Similar results were obtained when the animals were exposed to ether vapour or crushed ice. Leptazol (10 mM/10 min) induced 'convulsions' (strongly increased motor activity) associated with a reduction in the nerve cord ACh levels (Table 1). Eserine (0.2 mM/40 min) produced marked, sustained muscular contractions and an increase in ACh content of the nerve cord (Table 1).

Changes in ACh content in the leech nerve cord were similar to those in the mammalian brain after the administration of stimulant or anticholinesterase drugs (Pepeu & Nistri, 1973). However, during anaesthesia mammalian brain ACh content is increased (Pepeu & Nistri, 1973) whereas in the leech nerve cord there was no change in ACh content.

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Anticonvulsive action of homotaurine and taurine

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The anticonvulsive actions of homotaurine and taurine were tested in cats and rats in comparison with γ-aminobutyric acid (GABA) and glycine. In addition, the cortical levels of taurine and homotaurine were tested, at the end of each experiment, by chromatographic techniques (Guidotti, Badiani & Pepey, 1972).

Acute midpontine brainstem transected cats were made epileptic: (a) by local application, on the left and the right sensorimotor cortices, of either cobalt powder (50 mg) or a physostigmine Ringer solution (1.10^{-4}); or (b) by i.v. perfusion of a strychnine sulphate solution (0.5 mg/ml) in curarized cats. The EEG and arterial BP were always recorded.

After the appearance of the epileptic seizures of both cerebral hemispheres, a solution in saline 0.3% of the aminoacid under investigation was slowly (25 mg/min) perfused through the right lingual artery in the circulation of the right cerebral hemisphere.

The appearance of an EEG asymmetry between the right and the left cortices was considered to be the effect of the aminoacid perfusion.

Under these experimental conditions GABA and glycine (up to 1 g) showed no protective

Table 1 Time of convulsion evocation by hyperbaric oxygen

Rat breed	n	*Saline*	*GABA (370 mg/kg i.p.) three times; once every 12 h*	*Homotaurine (500 mg/kg i.p.) three times; once every 12 h*
Wistar	5	476 ± 39*	620 ± 52	734 ± 54*
Sprague Dalley	5	788 ± 46** †	1010 ± 57**	927 ± 22†

* $P < 0.01$; ** $P < 0.02$; † $P < 0.05$.

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effect against epileptic seizures however they were evoked. On the contrary taurine and homotaurine were effective in the protection against the epileptic seizures evoked by cobalt, but were not effective against the physostigmine and strychnine evoked convulsions.

Nevertheless while taurine (0.5 g–3 experiments) had practically no protective action on the epileptic focus and was only partially effective on the diffusion of the seizure activity, homotaurine, at equimolecular doses (3 experiments), was totally effective against cobalt evoked seizures of both hemispheres. Furthermore the homotaurine perfusion caused a synchronized EEG tracing.

A single i.p. injection (100 mg/kg) of taurine or homotaurine, did not protect rats against convulsions due to hyperbaric oxygen, nevertheless, as shown in Table 1, homotaurine (500 mg/kg) given i.p. three times at 12 h intervals, significantly protected rats against hyperbaric oxygen convulsions.

In conclusion our results confirm the anticonvulsive action of taurine (Barbeau & Donaldson, 1973; Van Gelder, 1972) and show that homotaurine is considerably more potent than taurine in preventing epileptic seizures evoked by cobalt and hyperbaric oxygen.

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Brain levels of the potent analgesic etorphine in rats and their functional significance

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Pharmacological data and *in vitro* binding studies suggest the existence of specific opiate receptors. However, *in vivo* measurements of opiate narcotics in the CNS in general fail to correlate with the functional condition (e.g. analgesia, tolerance, dependence). Such a lack of correlation most probably results from the diffuse distribution of these compounds in tissues, with only a minor drug fraction attached to specific receptors.

With potent narcotics that are effective in very small doses and low total tissue concentration, the ratio between the amount of drug at receptor sites and the latter may be more favourable. Indeed this is somewhat substantiated with the potent morphine-like drug etorphine labelled with tritium ([^{3}H]-ET) in the work of Dobbs (1968), that we have confirmed and expanded using more reliable techniques (i.v. administration of [^{3}H]-ET and identification of [^{3}H]-ET in tissue by TLC).

Table 1 Effect of cyprenorphine on [^{3}H]-etorphine levels in rat brain

Expt. no.	Treatment (μg/kg) [^{3}H]-etorphine (i.v.)	Cyprenorphine	Brain levels of etorphine 15 min after injection (ng/g ± s.d.)	No. of animals
I	2	–	0.69 ± 0.03	4
	2	100 (i.p. 30 min before)	0.43 ± 0.09	4
II	20	–	8.87 ± 1.54	3
	20	100 (i.p. 30 min before)	5.89 ± 0.49	3
	20	400 (i.p. 30 min before)	3.38 ± 0.23	3
III	20	–	9.96 ± 2.78	4
	20	100 (i.v. 5 min after)	4.40 ± 1.34	4

Tritium labelled (15 and 16 position) etorphine, S.A. 3.6 Ci/mM was injected into male Sprague Dawley rats (180-200 g). Analysis consisted of liquid scintillation radioassay of TLC purified acetone homogenates of individual brains. The means relative to cyprenorphine treated animals were all significantly different ($P < 0.01$) from those of corresponding controls, based on Student's t test (expt. no. I and III) or analysis of variance (expt. no. II).