When the effects of varying the stimulation frequency from 12·5–100 Hz upon the release of [^{3}H]noradrenaline from the caudate nucleus were examined, it was found that the initial period of stimulation at all frequencies tested in four experiments caused a significant increase ($P < 0.05$) in [^{3}H]noradrenaline perfusate concentration with the greatest release occurring at 50 Hz [increased release of [^{3}H]noradrenaline (nCi/ml) at 12·5 Hz = 0·8 $\pm$ 0·4, 25 Hz = 3·7 $\pm$ 1·6, 50 Hz = 6 $\pm$ 2·1, 100 Hz = 4 $\pm$ 0·7].

These facts support the idea of neurotransmitter roles for brain catecholamines by demonstrating depolarization-evoked release of [^{3}H]noradrenaline; the release is frequency-related, suggesting that it is related to neuronal function.

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Inhibitory action of γ-aminobutyric acid on cryoepilepsy in the frog

The inhibitory action of α-aminobutyric acid (GABA) in the central nervous system synapses has led some authors to investigate its influence on experimental convulsions and correlate its brain content in the central nervous system with convulsive disorders observed (Meynert & Kaji, 1962; Wood & Watson, 1969; Saad, 1970).

We report the action of GABA on an epileptiform attack produced by sudden cooling of the spinal cord of the frog (cryoepilepsy) (Ozorio de Almeida, 1943) and the relation to its content in the nervous centres.

GABA (0·5–5·0 g/kg) was injected into the ventral lymphatic sac of the frog and 1–48 h later the spinal cord was isolated and plunged into a temperature-controlled cooled Ringer bath (Ozorio de Almeida, Moussatché & Vianna Dias, 1941). After the induced convulsive attack, the cords were weighed, homogenized in 1 ml of ice-cold Ringer and centrifuged 15 min at 0° and 15 000 *g*. Free GABA was quantitatively estimated in the supernatant fluid by the *Ascaris lumbricoides* muscle bioassay (Ash & Tucker, 1967, as modified by Moussatché & Cordeiro, unpublished).

The relation between dose, temperature and the inhibition of convulsions is seen in Table 1. Doses of GABA greater than 3·0 g/kg, injected 1–5 h previously, inhibited the convulsions completely when the spinal cord was cooled to 6°; all the controls convulsed. The per cent inhibition of the convulsions is related to the bath temperature (Moussatché & Cuadra, 1967). After 24 or 48 h cords from GABA-treated frogs showed respectively 50%, inhibition or no inhibition when plunged in a bath at 5–6°.

Table 1. *The relation between temperature to which the spinal cord of frogs was exposed and the inhibition by GABA of epileptiform convulsions.*

GABA g/kg	Bath temperature °C	Convulsions observed %
3·0	4–6	0 (4)
5·0	4–6	9·5 (21)
5·0	0–4	33·3 (3)
Controls	4–7	100·0 (20)

Figures in brackets are number of frogs.

Table 2. *Concentration level of GABA in the spinal cord of the frogs and inhibition of cryoepilepsy at 5–6° C.*

	Time elapsed after GABA injection (5 g/kg) 1 h	3–5 h	24 h	48 h	Controls
GABA content	6·4±1·8*	4·5±1·0	3·1±0·2	1·4±0·2	3·0±0·4
Inhibition of convulsions (%)	100†	85†	50	0	0

* Each figure is the mean and s.d. of 3 groups of 2 frogs each.
† Different from controls $P < 0.05$.

In spite of the high dose of GABA injected and inhibition of the convulsions, the frogs themselves did not show apparent behavioural changes and jumped normally and showed normal postural reflexes.

Table 2 shows the GABA content in the spinal cord of the frogs and the per cent inhibition of the convulsions at intervals after GABA injection.

The results suggest that there is some relation between the concentration of GABA in the spinal cord of the frogs and the per cent inhibition of cryoepilepsy. These results are further evidence of the pharmacological activity of this amino-acid in the central nervous system.

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