we could often antagonise GABA evoked inhibitions by the simultaneous application of ACTH (13 out of 21 tests). Conversely, CDP often potentiated GABA evoked inhibitions of the same neurone (5 out of 10 tests). Perhaps the most interesting observation is that we have been able to reproduce the neurochemical evidence of antagonism between CDP and ACTH. Thus we were able to reversibly block ACTH evoked inhibitions by the simultaneous application of CDP (4 out of 6 tests).

Using two different pharmacological techniques we have been able to corroborate the earlier behavioural data showing an antagonism between ACTH and benzodiazepines. Thus, if ACTH is an endogenous anxiogenic compound then a functional antagonism with benzodiazapines would provide one possible clue to the mode of action of anxiolytic agents.

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Inactivation of released norepinephrine in rat tail artery by neuronal uptake

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Neuronal uptake of endogenously released norepinephrine plays an important role in transmitter disposition in vascular smooth muscle. Recent evidence (Verbeuren, Janssens & Vanhoutte, 1978) suggests that this neuronal pump operates mainly between nerve impulses and is inactivated during depolarization of the nerve endings. This allows for optimal diffusion of transmitter. If this reasoning is correct, an inhibitor of neuronal reuptake, such as cocaine, should differentially alter the contractile response to low and high frequency stimulation. This hypothesis was tested in the present study by comparing the contractile responses of rat tail artery strips to electrical stimulation in the presence and absence of cocaine. Adult male albino rats were killed by a blow to the head and tail arteries were obtained. The artery was cut helically into strips; the strips were mounted in organ baths between two platinum wire electrodes; and isometric contractions were recorded. Cumulative addition of cocaine to the bath produced contraction of the strips. This contractile response was blocked by phentolamine (10 μM) and was dependent upon the concentration of cocaine (10^{-8} to 10^{-4}M). Acute denervation of the strips with 6-hydroxydopamine (10 min, pH 4.0) reduced the contractile effect of cocaine. Contractions in response to low frequency field stimulation (0.1 to 1.0 Hz) were significantly potentiated by cocaine (10^{-6}M). Those in response to high frequency stimulation (2 to 16 Hz) were not significantly altered by the presence of cocaine. Cocaine produced a marked slowing of relaxation in tail artery strips contracted by field stimulation (1 and 16 Hz). This effect was concentration dependent (10^{-8} to 10^{-4}M); the relative magnitude of the interval of time required to reach half maximal relaxation was not different for low or high frequency stimulation. These results indicate: (1) contraction in response to cocaine alone probably results from the inhibition of uptake of spontaneously released norepinephrine; (2) the amine uptake mechanism in rat tail artery is differentially affected at low and high frequency electrical stimulation suggesting that neuronal uptake is not operative during the nerve impulse; and (3) the neuronal uptake of transmitter following electrical stimulation is an important disposition mechanism in rat tail artery.

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Could harmaline generate tremor by affecting 5HT release?

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Whole body tremor can be generated by rhythmical and synchronous activity in the inferior olivary nucleus and such activity can be induced by harmaline, presumably by modification of electrotonic