PHARMACOLOGY

EFFECT OF MORPHINE AND ITS ETHERS ON THE HYPERKINESIS CAUSED BY BARBITURATES

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The hyperkinesis arising in animals after the administration of sub-narcotic doses of barbiturates and evidenced by rapid contractions of separate muscle groups is of a cholinergic nature. This is confirmed by the fact that substances which affect the processes of cholinergic stimulation (acetylcholine, arecoline, carbachol, proserine, atropine, scopolamine) are able to change the force, the duration of barbituric hyperkinesis and even to suppress or prevent it [2].

We carried out a test of morphine and its ethers, which also act on the processes of cholinergic stimulation.

The experiments were carried out on mice which were injected subcutaneously with doses which did not provoke tail twitching (the so-called Straub reaction), namely 1-3 γ morphine, 0.5 γ heroine, 25-50 γ codeine per 1 g of weight. In 10-15 minutes, 60-80 γ/g of hexenal were administered to the mice.

These doses of hexenal caused clear and sufficiently prolonged hyperkinesis in the control animals. Mice receiving morphine or its ethers in advance not only experienced no hyperkinesis, but also no stage of locomotion stimulation which arises in animals after the administration of barbiturates and other narcotic drugs.

Thus, morphine and its ethers, which have little or no effect on convulsions of other origin, depress barbituric hyperkinesis, indicating the specificity of this action and making it possible to utilize it as an indicator of the effects of morphine.

According to published data [1, 3] the stimulation which leads to barbituric hyperkinesis is localized in the subcortical structures (the mesencephalon in frogs and, possibly, the diencephalon in mammals). We were discovering by what routes morphine affects this stimulation, acting on the cortex or on the subcortical centers. Experiments were undertaken in which morphine was administered to rats which had had the cerebral cortex removed earlier (from a month to a year in advance). In such animals (15 rats) barbituric (hexenal) hyperkinesis was well evident; when 0.5-2 mg of morphine per 1 kg of weight (doses which did not cause tail twitching) was administered earlier, hyperkinesis was clearly evident only in 2 animals, very weakly evidenced in six, and completely absent in 7 out of 15 animals. These results indicate that morphine depression of barbituric hyperkinesis depends primarily on the effect of morphine on the subcortical structures.

LITERATURE CITED

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