As shown in Table 1, the potency of chlorprothixene was the strongest; perphenazine was second; next in order were levomepromazine, chlorpromazine, TX-102, Pasotomin and Psytomin. It was found that chlorpromazine and perphenazine were in a group in terms of slop function, and that levomepromazine, chlorpromazine, TX-102 and Pasotomin were in another one. Only Psytomin was far from both groups. From Tables 1 and 2, these antagonists showed the protective action against nicotine-induced death in hypnotic doses. The minimal protective action of these antagonists also appeared in sedative doses.

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ACCUMULATION OF ^{14}C -NICOTINE IN MOUSE BRAIN AFTER THE INTRAPERITONEAL INJECTION

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We have reported that pretreatment with small doses of reserpine (less than 0.5 mg/kg) had no effect on the level of nicotine in the brain and that of large doses decreased the level following intraperitoneal injection of convulsive dose of nicotine, though pretreatment with the small dose of reserpine inhibited nicotine-induced convulsion and that with the large dose accelerated this convulsion (1, 2). Thereafter we have examined the effect of reserpine on the accumulation of nicotine in brain particulate fractions especially in microsomes and nerve endings, but we could not find any reserpine effect on the accumulation *in vivo*.

TABLE 1. Accumulation of ^{14}C -nicotine in brain.

Time after ^{14}C -nicotine	Fractions	
	Nuclear	Soluble
5 (min)	123 (cpm)	2295
15	98	2040

FIG. 1. Accumulation of ^{14}C -nicotine in brain. MS; microsomes + myelin. NE; nerve endings. MT; mitochondria. Curves 1 and 2; 5 and 15 minutes after ^{14}C -nicotine respectively.

TABLE 2. Relative values of accumulated ^{14}C -nicotine per protein weight.

Time after ^{14}C -nicotine	Fractions		
	Microsomal	Nerve ending	Mitochondrial
5 (min)	12.8	8.4	0.5
15	10.5	8.5	2.7

the animals were killed by a blow. Brain cell fractions were separated according to the method of Whittaker (3) using density gradient centrifugation. In this procedure the particulate fractions were washed three times. ^{14}C -nicotine in each fraction was extracted and determined by the method as described elsewhere (1). Protein concentration was assayed by the method of Lowry *et al.* (4).

As shown in Table 1 and Fig. 1 the greater part of radioactivity was in soluble fraction. Less than one-tenth of radioactivity of soluble fraction was found in microsomes (plus myelin) and nerve endings. The radioactivity in microsomes was somewhat higher than that in nerve endings. The maximal levels of nicotine in brain cortex and stem have been shown 5 to 10 minutes after nicotine, and the levels decreased in two-thirds after 30 minutes (1). In the present findings the accumulation of nicotine after 5 minutes was more or less higher than after 15 minutes in all fractions but mitochondria. Relative value of accumulated radioactivity per protein weight in microsomes was higher than those in nerve endings and mitochondria, and the accumulation and the relative value in mitochondria were the least in the three particulate fractions (Table 2).

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CHANGES OF BRAIN CALCIUM CONTENT IN NORMAL AND MORPHINIZED MICE AFTER THE ADMINISTRATION OF MORPHINE

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We have reported that the analgesic effect of morphine given subcutaneously or intracisternally were markedly suppressed by the intracisternal injection of calciumion and potentiated by EDTA (1, 2).

On the other hand, *in vitro* experiments, the inhibition of potassium stimulated oxygen uptake of cerebral cortical slices by morphine could be observed only in the low calcium media and that in the cerebral cortical slices from normal rats but not from rats tolerant to morphine (3).

As a part of our present investigation concerning the significance of calcium in morphine analgesia, we studied the changes of brain calcium content after the administration of morphine in normal