hydrochloride were without effect in doses of 400-1000 γ. Nicotine base exerted a partial effect in 100 γ dose; histamine was effective only in toxic amounts of 400 γ and acetylcholine chloride was ineffective in a dose of 50 γ. This analysis shows that although the several quarternary alkaloids and other drugs mentioned in Table II may influence the synaptic transmission or the myoneural junction, they do not compare with curare and erythroidine in their influence on the outward manifestion of morphine poisoning in mice. It may be noted here that Myanesin[9] [α,β-dihydroxy-γ-2-(methylphenoxy-propane)] in doses of 8 mg per mouse despite its curare-like action on the striated muscles, is not able to depress the tail reflex in morphinized mice. The data seem to indicate that because of the relatively small amounts of alkaloid required to produce a positive effect, this method may be useful for the determination of curare or erythroidine in tissue extracts or urine even though other active substances may be present.

Summary. A biological method for qualitative and quantitative determination of curare and erythroidine is described. The method depends upon the antagonistic action of these alkaloids on the excitement and tail phenomenon in morphine-poisoned mice. The median effective dose and standard error for crystalline *d*-tubocurarine chloride is 2.8 ± 0.2 μg; for Strychnos Curare (Merck), 24.0 ± 2.0 μg; for dihydro-β-erythroidine bromide, 44.0 ± 3.0 μg; and for Intocostrin (Squibb), 20.0 ± 2.0 milliunits. Far higher doses of other drugs, including quarternary alkaloids, are necessary to antagonize the effect of small amounts of morphine in mice.

9 Berger, F. M., and Bradley, W., *Brit. J. Pharmacol.*, 1946, **1**, 265.

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Effect of Anoxic Anoxia on Propulsive Motility of the Small Intestine.*

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It has been shown that although anoxic anoxia does not significantly affect the propulsive motility of the small intestine of the dog, it does so in the mouse.[1] Since there is this apparent specific difference it was deemed worthwhile to study the effect of anoxic anoxia on still a different species, namely, the rat.

Methods. Paired albino rats as nearly alike in weight and age as possible were fasted 24 hours. One served as a control and the other was subjected to anoxic anoxia. (In all, 42 control and 42 experimental animals were used.) The following partial pressures of oxygen were employed: 94 mm Hg, 80 mm Hg, 63 mm Hg, and 53 mm Hg; corresponding to simulated altitudes of 14,000, 18,000, 24,000, and 28,000 feet.

The experimental animal was given 2 cc of a charcoal-acacia mixture by stomach tube, and after allowing 10 minutes for some of this mixture to enter the small intestine, placed into a low-pressure chamber for 30 minutes. On removal from the chamber it was decapitated and the small intestine removed. The latter was slit open and the distance the charcoal-mixture had traversed the small intestine was measured. Control intubated animals were maintained at atmospheric pressure; in order to keep other experimental conditions as nearly alike as possible, they were placed in cages on top of the low-pressure chamber, so that they were exposed to the same noise and vibration as the experi-

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1 Van Liere, E. J., Northup, D. W., Stickney, J. C., and Emerson, G. A., *Am. J. Physiol.*, 1943, **140**, 119.

TABLE I.
Effect of Anoxia on Peristalsis of the Small Intestine in Rats.

PO_2 mm Hg	Altitude, feet	Control		Anoxia		"P"*
		No. of animals	% of gut traversed at end of 40 min.	No. of animals	% of gut traversed at end of 40 min.	
94	14,000	11	71	11	64	0.19
80	18,000	9	82	11	64	<0.01
63	24,000	10	76	10	50	<0.001
53	28,000	12	78	10	40	<0.001

* Significant when "P" (according to Fisher) is 0.05 or less.

mental animals.

Results and Discussion. The data, Table I, show that anoxic anoxia did not decrease the propulsive motility of the small intestine significantly at a simulated altitude of 14,000 feet. Above this level, however, the motility was significantly decreased by the anoxia.

The results indicated that the threshold for anoxia on the peristalsis of the small intestine lay between a simulated altitude of 14,000 feet and 18,000 feet. In previous work[1] it was shown that the propulsive motility of the intestine of the mouse was not significantly decreased until a simulated altitude of 18,000 feet was reached. The results obtained with rats, therefore, show fair agreement with those for mice. Since anoxic anoxia stimulates the sympathetic division of the autonomic nervous system it would be expected that intestinal movements would be decreased.

The authors still can offer no adequate explanation for the negative effect of anoxic anoxia on the propulsive motility of the small intestine in the dog. The current experiments suggest that as far as the propulsive motility of the small intestine is concerned the rat is somewhat less resistant to anoxia than the dog. However, it should be pointed out that both of these animals are relatively resistant to anoxic anoxia so that the difference observed by us between the two species probably cannot be attributed entirely to this factor.

Summary. The effect of anoxic anoxia on the propulsive motility of the small intestine of the albino rat was studied at the following partial pressures of oxygen: 94 mm Hg, 80 mm Hg, 63 mm Hg, and 53 mm Hg; corresponding to simulated altitudes of 14,000, 18,000, 24,000, and 28,000 feet. At a simulated altitude of 18,000 feet or higher there was a significant decrease in propulsive motility. The threshold lay between 14,000 and 18,000 feet. The results differ from those obtained in the dog but show fair agreement with those in the mouse.

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Protein Level and Pteroylglutamic Acid Intake on Growth Rate and Hemoglobin Level in the Rat.

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In attempts to produce a pteroylglutamic acid (PGA) deficiency in the pig, baby pigs were fed a PGA-low "synthetic" diet containing 30% casein and 2% sulfathalidine. However, no blood dyscrasias were observed.[1] Kornberg, Daft and Sebrell[2] and Daft[3] have shown that limitation of the casein in the

1 Johnson, B., Connor, James, Marian, F., and Krider, J. L., *J. Animal Science*, 1947, **6**, 486.

2 Kornberg, Arthur, Daft, Floyd S., and Sebrell, W. H., *Science*, 1946, **103**, 646.

3 Daft, Floyd S., *Fed. Proc.*, 1947, **6**, 405.