TABLE I.
Swing Tests on Subjects After Various Substances.

		Drug		Placebo			
	mg	No. Tested	No. Vomited	No. Tested	No. Vomited	Estimated protection %	P§
Barbital, sodium	325	20	12	20	9	—	1.00
Benzedrine sulfate	10	20	13	20*	14	7	0.73
V-5	250	21	13	21*	15	13	0.52
Thiamine chloride	10	20	12	20*	14	14	0.66
USAMSP		19	11	19*	16	31	0.07
Sulfadiazine, 1 g†		19	4	18	4	0	1.00
Sulfadiazine, 1 g‡		19	5	17	7	36	0.33
Hyoscine hydrobromide	0.75						
prostigmine bromide	15.0	20	10	21	20	48	0.01
Hyoscine hydrobromide	0.75						
prostigmine bromide	15.0	81	17	365	98	22	0.28
Pyridoxine hydrochloride	100	33	15	82	30	—	1.00
Pyridoxine hydrochloride	200	28	9	82	30	12	0.66
Hyoscine hydrobromide	0.75						
V-12	250	104	14	365	98	50	0.01
Hyoscine hydrobromide	0.75						
chlorbutanol	500						
benzedrine sulfate	10	145	14	365	98	64	0.01

* Subjects acted as own controls, receiving the drug the second or third time swung and the placebo the other time.
† Tested after receiving the drug for 4 weeks.
‡ Tested after receiving the drug for 7 weeks.
§ Probability that observed difference could have occurred by chance.

ficant.

Summary. 1. Sodium barbital, benzedrine. V-5, thiamine, and pyridoxine were of no demonstrated value in the prevention of swing sickness. 2. Sulfadiazine in daily doses of 1 g did not appreciably affect the susceptibility to swing sickness. 3. A mixture of sodium amytal, atropine, and scopolamine was effective in swing sickness but probably not significantly more effective than could be predicted from its content of atropine and scopolamine. 4. The addition of neostigmine to scopolamine decreased the effectiveness of this mixture over that of scopolamine alone but did not entirely abolish it, although it effectively prevented the depressant action of scopolamine on salivation. 5. The addition of the thiobarbiturate V-12 to scopolamine did not increase its effectiveness in swing sickness. 6. A mixture of scopolamine, chlorobutanol and benzedrine was insignificantly more effective than the scopolamine alone in a similar dose.

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Effect of Anoxic Anoxia on Body Weight Loss in Rats.

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Fasting rats lose a significantly greater amount of weight when exposed to simulated altitudes of 8,000, 18,000 and 28,000 ft. than at normal barometric pressure.[1,2] Since the effect of anoxia on the weight and the water

[1] Van Liere, E. J., *Anoxia, Its Effect on the Body*, The University of Chicago Press, Chicago, 1942.

[2] Swann, H. G., and Collings, W. D., *J. Aviation Med.*, 1943, **14**, 114.

total loss, "net loss" and feces were between 4,000 and 8,000 ft.; for urine below 4,000 ft.

The curves for per cent increase in total weight loss and "net loss" for the 2 groups of rats are fairly consistent with each other and together indicate increases proportionate to the altitude. The rats of Group 4 did not display as intense a polyuria in response to anoxia as those of Group 1, but they, nevertheless, had an increase in urine secretion which was proportionate to the altitude. The rats of Group 4 had a greater increase in fecal excretion at all altitudes than those of Group 1. These differences between the 2 groups were most probably due to the character of the diet; that for Group 1 being inferior. The eventual decline of the fecal excretion curves with altitude was probably due to a reduced intake of the diet, since loss of appetite is a well known symptom of anoxia.[1] The increased fecal excretion confirms the impression gained in this laboratory that in dogs, also, defecation is frequently one of the responses seen at 28,000 ft. It is of interest that colonic motility as registered by an enterograph in barbitalized dogs is usually greatly reduced by anoxia.[4]

Swann and Collings,[2] who subjected their rats under different conditions to 18,000 ft. for periods of time between 6 and 23 hours, found much greater increases in the rate of insensible water loss than reported here. This may explain in part their failure to find a diuresis at high altitude. Time may be a factor also, because their results for an exposure of 23 hours were less than those at shorter exposures and practically no different than the controls. Their finding an increase in the rate of fecal loss during the 6-hour exposure only, is to be expected in fasting rats.

The increases, here reported, in urine secretion are even greater than those found by Silvette.[5] The few experiments in which cocaine was administered did not reveal increased urine secretion above that seen without this epinephrine-potentiating agent, nor did they reveal a reduced secretion. This result was obtained in spite of the fact that cocaine caused in the control experiments, an increase of over 200% in urine secretion. Such results are not favorable to the view that anoxia-induced changes in urine secretion are a result alone of epinephrine and/or augmented sympathetic impulses to the kidneys. Such an explanation is more in accord with the results found in anesthetized animals[6] where oliguria is frequently found as well as polyuria in response to anoxia. Furthermore, 32,000 ft. proved lethal to all but one of the rats, but in spite of this severe stimulation of the autonomic nervous system, they continued to respond with polyuria.

Summary. The body weight loss in rats is proportional to the altitude up to 28,000 ft. The thresholds for increased total body weight loss, "net loss" (insensible water loss) and fecal excretion lie between 4,000 and 8,000 ft.; that for urine, below 4,000 ft. The administration of cocaine did not change the urinary response to anoxia (28,000 ft.)

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

[5] Silvette, H., *Am. J. Physiol.*, 1943, **140**, 374.

[6] Toth, L. A., *Am. J. Physiol.*, 1940, **129**, 532.

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Neural Effects of DDT Poisoning in Cats.

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The literature of the last few years contains several reports[1,2] of nervous disorders produced in laboratory animals by DDT poisoning. Our aim was to investigate whether DDT has, to any extent, a selective action

[1] Lillie, R. D., and Smith, M. I., *Pub. Health Rep.*, 1944, **59**, 979.

[2] Nelson, A. A., Draize, J. H., Woodard, G., Fitzhugh, O. G., Smith, R. B., Jr., and Calvery, H. O., *Pub. Health Rep.*, 1944, **59**, 1009.