We wish to offer the *suggestion* that impulses constantly pass from various organs to the vomiting center and that apomorphin promotes the coördinated vomiting reflex to such a degree that these normal impulses give rise to vomiting.

That the action of apomorphin on the vomiting center is strictly analogous to that of strychnin on the cord whereby convulsions—apparently spontaneous, but in reality of reflex character—are induced.

Vomiting requires powerful—almost *convulsive*—contractions of the abdominal muscles and diaphragm, and the weak stimuli are incapable of setting up the reflex in the unpoisoned animal.

It is significant, too, that morphin produces strychnin-like convulsions through its action on the cord (in the frog), and apomorphin-like emesis through its action on the medulla.

5 (1752)

III. Experimental rickets.

The prevention of rickets in rats by exposure to sunlight.[1]

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In recent papers it was shown by Hess and Unger that rickets in infants could be cured by frequent short exposures to the sun's rays.[1, 2] By this means and without any alteration whatsoever of the dietary, the characteristic signs of this disorder begin to disappear in three to four weeks as noted by clinical examination and by the x-ray. As a result of favorable experiences of this nature, it was concluded in a study of the seasonal incidence of rickets[3] that "hygienic factors, especially sunlight, and not dietetic factors play the dominant rôle in the marked seasonal variations of this disorder." It seems probable that the ultra-violet rays play a large part in this curative power of the sun, judging from the work of Huldschinsky[4] and others who recently have shown that

[1] Hess, A. F., and Unger, L. J., PROC. SOC. EXPER. BIOL. AND MED., 1921, xviii, 298.

[2] Hess, A. F., and Unger, L. J., *J. A. M. A.*, 1921, lxxvii.

[3] Hess, A. F., and Unger, L. J., *Amer. J. Dis. Child.*, 1921, xxii, 186.

[4] Huldschinsky, K., *Zeitschr. f. orthop. Chir.*, 1920, xxxix.

infantile rickets can be cured by means of the rays produced by the mercury-vapor lamp. In 1918 we tried the curative effect of rays from this source, but, lacking the aid of x-ray examinations, could not convince ourselves of their efficacy; since then we have succeeded in curing rickets by this means.

Having found sunlight efficacious in the rickets of infants, we proceeded to test its value in the prevention of rickets in rats. To this end a series of white rats were placed on the diet (No. 84) described by Sherman and Pappenheimer,[5] consisting of patent flour 95.0 per cent., calcium lactate 2.97 per cent., sodium chloride 2.0 per cent. and ferric citrate 0.1 per cent. It has been the experience of the investigators in this laboratory that such a diet invariably leads to the development in rats of lesions which are anatomically identical with those of infantile rickets.

In carrying out experiments on rats our practice has been to keep the colony in a semi-dark room, the yellow shades being drawn at all times. In testing the effect of sunlight, the rats (weighing at the outset about 40 grams) were kept in absolute darkness, one series being taken out of the room and exposed to the direct sunlight for a period of 15 or 30 minutes. There was no difference whatsoever in the diets of these two groups. After a period of about three weeks the animals were x-rayed in order to observe early lesions of the epiphyses, and after thirty to forty days were killed and autopsied. These experiments were begun in April when the weather permitted four to five exposures a week.

It was found for the first time in our experience that diet No. 84, the "rachitic dietary," did not lead to rickets—that the rats which received sun treatment did not show signs of rickets either by x-ray or by histological examination of the bones. It is unnecessary to discuss in detail the histological criteria which we consider characteristic of rickets, as this question has been fully considered in a previous paper.[6] It may be stated briefly that they consist of increased width and irregularity of the proliferative cartilage, absence of calcium deposition and great excess of

[5] Sherman, H. C., and Pappenheimer, A. M., *J. Exper. Med.*, 1921, xxxiv, 189.

[6] Hess, A. F., McCann, G. F., and Pappenheimer, A. M., *J. Biol. Chem.*, 1921, xlvii, 395.

osteoid in the region of the metaphysis and along the shafts of the bones.

In the paper previously referred to it was shown that the introduction of 0.4 per cent. secondary potassium phosphate (K_2HPO_4) in place of an equal weight (replacing about one seventh) of the calcium lactate contained in the rickets-producing diet, completely prevented the development of rachitic lesions; this constitutes an addition of 75 mg. of phosphorus per 100 gm. of the diet. In order to test the counterbalancing effect of phosphate and darkness, a series of tests were carried out in the dark with additions of small and increasing amounts of potassium phosphate to the standard dietary (No. 84); to one series 25 mg. were added, to another 75 mg. (constituting dietary No. 85).

The rats on these diets were kept in the dark but, to serve as control, half of each series were exposed to sunlight for thirty minutes daily when this was possible. As was to be expected in view of our previous experience and the fact that phosphate tends to protect against rickets, none of the rats which were treated with sunlight developed rachitic lesions. Among the group, however, which were kept at all times in the dark, active rickets developed in spite of an addition of 25 mg. of phosphorus. The addition of 75 mg. was found to be sufficient to prevent the development of this disorder in some of the rats. This amount contituted somewhat less than the minimum protective supplement to diet No. 84, which in itself contains about 86 mg. of phosphorus. Thus it will be noted that a short exposure to sunlight was equivalent to almost doubling the protective dose of phosphate. If the phosphate content of the diet is adequate, rats do not develop rickets in spite of being kept in the dark throughout the experiment. The effect of sunlight with other dietaries was also studied, and is being continued.

DISCUSSION.

As sunlight has a marked effect on the bony development of rats, it is evident that in future in similar nutritional investigations, the light factor will have to be controlled and standardized. It seems probable that some of the irregularities and lack of conformity observed by investigators in this field may be attributed to keeping the experimental animals under dissimilar intensities of

A. DARKNESS.

Diet.[1]	Duration (Days).	Rat No.	X-Ray.	Microscopic Examination.
No. 84.....	34	246	R.	R.
	23	247	—	R.
	22	248	—	R.
	—	436	R.	—
	—	437	R.	—
	30	438	R.	R.
No. 84 + 25 mg. P.....	39	262	R.	R.
	39	263	R.	R.
	39	264	R.	R.
	28	443	R.	—
	28	444	R.	R. (slight)
	28	445	R.	—
No. 84 + 75 mg. P.....	38	121	neg.	neg.
	38	122	neg.	neg.
	38	123	neg.	neg.

B. SUNLIGHT.

Diet.	Duration (Days).	Rat No.	X-Ray.	Microscopic Examination.
No. 84.....	34	249	neg.	neg.
	32	250	"	"
	35	251	"	"
	33	439	"	"
	33	440	"	"
	33	441	"	"
	33	442	"	"
No. 84 + 25 mg. P.....	39	259	"	"
	39	260	"	"
	39	261	"	"
No. 84 + 75 mg. P.....	38	124	"	"
	38	125	"	"

R. = rickets.

light. The most interesting aspect of the question, however, is the phenomenon that the sun's rays are able to stimulate a deposition of inorganic salts where these are lacking. The damaging effect of darkness emphasizes the fact that sunlight is of great impor-

[1] Diet No. 84 as originally constituted, contained 86 mg. per cent. of phosphorus. In the fall, however, owing to a variation in the phosphorus content of the flour, this diet was found by analysis to contain only 72 mg. per cent. Rats numbered our 400 (in this table) were fed on the ration having the lower phosphorus content.

tance, not merely for the vegetable world but also for the higher animals. Furthermore, the fact that sunlight is efficacious in the rickets of both human beings and rats, serves to show the similarity of this disorder in these two species. These results indicate that in the prevention and causation of rickets at least one hygienic factor plays an important rôle which will have to be carefully considered in future studies of this disorder.

6 (1753)

Identical twins in pigeons arise from ova of markedly aberrant size.

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During 10 years data have been accumulated for yolk size and total egg size in 15,000 to 18,000 eggs of doves and pigeons. Such measurements of these two associated structures permit us, within certain limits, to know some definite things concerning the size of either structure if the weight of the other is known. Another group of 15,000 to 20,000 eggs have been weighed, incubated, and later observations made upon the embryos and young. Incidental to these latter observations 7 instances of identical twins have been found. Such twins other than the seven listed here have almost certainly not appeared; or, if present, they attained a stage of less than 2-day embryos.

The figures of Table I make it clear that at least most of the particular eggs which gave rise to twins were of markedly different size from all other eggs then being produced. This is particularly well shown in the first four instances—given in the upper eight rows of figures—since the twin-bearing egg was in these four cases by far the *largest* egg produced by its parent during one entire year,—and so much larger as to indicate, in all probability, that it contained the largest ovum produced during the year. The seventh case was likewise of aberrant size—being the *smallest* of a group of undersized eggs. However, the weights of all eggs obtained in connection with this seventh case, as also with cases 5