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Further observations on the seat of the emetic action of the digitalis bodies.

By ROBERT A. HATCHER and SOMA WEISS.

[From the Department of Pharmacology, Cornell University Medical College, New York City.]

The application of digitalis bodies directly to the vomiting center, described by Thumas, does not cause emesis.

When a digitalis body is injected into a cat in which the spinal cord has been cut at about the level of the second thoracic vertebra, vomiting does not usually occur, but when the cord is severed at the level of the 5th thoracic vertebra vomiting is not prevented.

Removal of the stellate ganglia frequently prevents this emesis, and removal of the stellate ganglia with cutting of both vagi prevents the emesis in nearly every case.

Removal of the celiac plexuses does not interfere with the emesis following the injection of digitalis bodies.

When the nerve supply to the heart is intact the injection of a digitalis body causes emesis, if the animal is in good condition. When all nerve supply to the heart is severed, digitalis does not cause emesis, but mercuric chloride still causes vomiting in the usual way.

Impulses appear to pass up from the heart to the vomiting center chiefly by the way of the sympathetic, and to a less, though probably variable, extent by way of the vagus. When the sympathetic is cut the administration of ouabain *usually* fails to induce emesis. This may be due to the fact that the impulses passing up the vagus are usually insufficient to set up the coördinated reflex, or it may be that in those cases where vomiting is not elicited by this drug after the sympathetic is cut the vagus does not contain any fibers concerned in this reflex.

Evidence is presented to show that digitalis bodies induce emesis by reflex action due to irritation of the heart or its appendages.

This is almost certainly a protective mechanism for the heart such as is recognized in the case of other organs.

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.

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III. Experimental rickets.

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

By ALFRED F. HESS, L. J. UNGER and A. W. PAPPENHEIMER.

[From the Department of Pathology, College of Physicians and Surgeons, Columbia University, New York City.]

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 rickets³ 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 Huldchinsky⁴ and others who recently have shown that

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

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

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

⁴Huldchinsky, K., Zeitschr. f. orthop. Chir., 1920, xxxix.