TABLE I

MAXIMUM OBSERVED BOTTOM VELOCITIES AT VARIOUS LOCALITIES

Depth meters	Maximum velocity cm/sec.	Submarine topographic feature	Nature of bottom	Number of observations
42	7.3	La Jolla Canyon	Silty sand (thin cover)	14
58	10.6	Newport Canyon	Black mud	15
73	10.7	Santa Monica Shelf	Silty sand and rock	80
91	26.9	Monterey Canyon	Sandy silt (thin cover)	16
182	14.7	Scripps Canyon		6
235	20.4	La Jolla Canyon	Silty sand	31
375	36.7	Scarp off San Pedro hill	Sandy silt	6
560	23.0	La Jolla Canyon	Silty sand	6
759	8.8	Saddle near San Clemente Island	Glauconitic and calcareous sand	2
780	18.8	San Pedro Canyon		1
796	20.8	Coronado Canyon	Mud	4
840	8.0	San Pedro Canyon	Rock and sand	3
886	17.7	San Pedro Basin	Silty mud over sand	3

showed the absence of a velocity strong enough to make a record (less than 2 or 3 cms per second). Such periods were occasionally followed by some of the highest measured velocities. In the submarine canyons the observed directions of movement showed a tendency to follow the axes of the canyons, but shifts in direction from up to down canyon occurred at irregular intervals. The bottom currents thus appear to be non-tidal in character, although they exhibit some tidal components. Even on the continental shelf where tidal components might be expected to predominate, non-periodic changes in velocity were encountered.

The observed irregular movements of the bottom water probably can be best interpreted as indicating the presence of large moving eddies with vertical axes.[4] The presence of silts and muds on the bottom in certain areas of highest observed currents indicates that these eddy currents are not competent to prevent all deposition. Nevertheless, such currents must play an important part in the transportation of fine sediment along the sea floor. Since evenly distributed eddies can not alone produce any net transport, however, other factors such as the gravitational component down slope and residual currents must cooperate to prevent deposition on the many areas of hard bottom off the California coast. Possibly, also, the currents are not as competent to move debris as might be expected from observations on the transporting power of rivers, since it is probable that velocities decrease more rapidly near the sea bottom[5] than near the bottoms of rivers.[6]

These bottom currents may be looked on as part of a mechanism for carrying sedimentary material, brought into the ocean by floods or by wave erosion, out into considerable depths of water. This transporting ability, however, should not be thought of as equivalent to cutting power sufficient to erode great submarine canyons out of the rock of the ocean bottom.

Acknowledgements: The work described in this note was made possible through a grant from the Geological Society of America to Shepard and through the active cooperation of the Scripps Institution and the assistance of the Kerckhoff Marine Laboratory.

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INHIBITION OF GASTRIC SECRETION BY EXTRACTS OF NORMAL MALE URINE[1]

IT is a well-established fact that gastric secretion and motility are inhibited by the ingestion of fat. That the inhibition is mediated humorally was proved in 1926 by Farrell and Ivy,[2] who found that the oral administration of fat inhibits motor activity in the transplanted and denervated gastric pouch. Feng, Hou and Lim[3] subsequently demonstrated that fat also inhibits secretory activity of the transplanted gastric pouch. Quigley, Zettleman and Ivy[4] showed that sugars likewise inhibit gastric motility by a humoral mechanism. It has been demonstrated that the humoral agent is not fat or one of its products of digestion, nor a constituent of thoracic duct lymph. Bile and the duodenal hormones, secretin and cholecystokinin, have also been eliminated from consideration.[3, 4] Evidence that the humoral agent is a specific duodenal chalone was proved by Lim and his coworkers,[5] who, after finding that a preparation of duodenal mucosa provided by Ivy inhibited gastric secretion, successfully prepared extracts of the duodenal mucosa which inhibited gastric secretion and motility. The active principle was given the name enterogastrone. Gray, Bradley and Ivy[6] later prepared more potent extracts of enterogastrone and defined a tentative unit based on the degree of inhibition of gastric secretion in dogs.

Although we have previously believed that both motor and secretory inhibition were produced by one

[1] Aided in part by a grant from the Committee on Endocrinology of the National Research Council.

[2] J. I. Farrell and A. C. Ivy, *Am. Jour. Physiol.*, 76, 227, 1926.

[3] T. Feng, H. Hou and R. K. S. Lim, *Chin. Jour. Physiol.*, 3: 371, 1929.

[4] J. P. Quigley, H. J. Zettleman and A. C. Ivy, *Am. Jour. Physiol.*, 108: 643, 1934.

[5] T. Kosaka and R. K. S. Lim, *Chin. Jour. Physiol.*, 4: 213, 1930; *ibid.*, 7: 5, 1933; R. K. S. Lim, S. M. Ling and A. C. Liu, *ibid.*, 8: 219, 1934.

[6] J. S. Gray, W. B. Bradley and A. C. Ivy, *Am. Jour. Physiol.*, 118: 463, 1937.

[4] C.-G. Rossby, *Jour. Marine Research*, 1: 3, 239–262, 1938.

[5] Fleming and Revelle, *ibid.*

[6] W. W. Rubey, *U.S.G.S. Professional Paper* 189E, p. 132, 1938.

substance, we have recently discovered that wide variations in the ratio of secretory to motor inhibition may occur in preparations subjected to different methods of concentration. This suggests that enterogastrone may consist of two principles, one which inhibits secretion and one which inhibits motility. A difficulty in the biological assay has arisen from the fact that animals occasionally become refractory to enterogastrone preparations because of protein impurities.[7] For this reason we have not tried our preparation in the treatment of "peptic" ulcer.

Our attention has been recently directed to the urine as a possible source of enterogastrone uncontaminated by the protein impurities which are present in the mucosal extracts. Sandweiss, Saltstein and Farbman[8] recently reported that extracts of pregnancy urine (Antuitrin-S) containing the gonadotropic hormone, prolan, are potent in preventing the development of jejunal ulcers in dogs subjected to the Mann-Williamson operation. Culmer, Atkinson and Ivy[9] have administered extracts of pregnancy urine daily to Pavlov pouch dogs and observed a significant reduction in gastric secretion. Since the inhibition was evident on the first day, within one-half hour after the first injection of the extracts, it was concluded that the latter had a direct action on the gastric glands. Necheles has stated that he was able to extract from human urine a substance which inhibits gastric secretion.[10] At the San Francisco meeting of the American Medical Association, Sandweiss and associates reported that extracts of normal female urine are also potent in preventing experimental ulcers in dogs. This finding definitely excluded prolan as the active constituent, since this hormone is believed to originate in the placenta. Recently Sandweiss placed at our disposal a sample of this extract of normal female urine, which we found to be potent in inhibiting gastric secretion in dogs. Since the first possibility to be considered in explaining this observation is that enterogastrone is excreted in the urine, we have attempted to extract the chalone from this source.

We have found that extracts of normal male urine, prepared by benzoic acid adsorption or tannic acid precipitation,[6] are very potent in inhibiting gastric secretion. Three milligrams of solid material, representing approximately 150 cc of urine, contain one enterogastrone unit of activity. This material is approximately 16 times as potent as the usual material prepared from the duodenal mucosa of hogs. Two dogs which have become refractory to mucosal preparations have responded to the urine extracts. No inhibition of gastric motility has been obtained with as much as three milligrams of the urine preparations, so that either the substance acting on gastric motility is not eliminated in the urine or else the method employed does not recover it. An assay carried out on five immature rats has revealed that three mgs of the urine preparations exhibit no trace of gonadotropic activity. The active principle is not affected by boiling for five minutes.

Although both the chemical and biological behavior of the substance in urine resembles that of duodenal preparations of enterogastrone, we can not as yet state that the active constituent of urine is actually enterogastrone. Attempts to identify the principle are in progress.

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[7] J. S. Gray and E. Wieczorowski, *Proc. Soc. Exp. Biol. and Med.*, in press.

[8] D. J. Sandweiss, N. C. Saltstein and A. Farbman, *Am. Jour. Dig. Dis. Nut.*, 5: 24, 1938.

[9] C. Culmer, A. J. Atkinson and A. C. Ivy, in press.

[10] H. Necheles, *Proc. Am. Gastroenterol. Soc.*, May, 1938.

NICOTINIC ACID IN SWINE NUTRITION

IN November, 1938, a herd of pigs in northwestern Pennsylvania was reported to the agricultural extension specialists of the Pennsylvania State College as being sick and unthrifty. These pigs were farrowed in September and weighed from twenty to forty-five pounds. Out of a total of seventy-six pigs, forty had died during October and November. The surviving pigs had stopped growing, were without appetite and were affected with diarrhoea and a dermatitis on the body and ears which had the appearance of a heavy scurf. This condition developed while the pigs were being fed a ration of corn, oats, wheat middlings, tankage (34 per cent. protein) and a limited amount of skimmed milk. They had access to good grass pasture which had never previously been used for swine.

At the time the case was reported these pigs were accustomed to huddle in their pens rather than to take advantage of the available pasture. When food was placed before them they would come to the trough, eat a small amount and refuse the remainder of the feed.

The condition of these animals baffled the local and district veterinarians and the case was brought to the attention of the college swine specialist. It was then noted that the symptoms of the disorder were similar to those reported by Chick and co-workers,[1] with pigs suffering from nicotinic acid deficiency on a diet consisting largely of maize. As a result of this observation seven pigs, having been selected as being in the poorest condition of any in the herd, were given 50 mg daily of nicotinic acid mixed in a minimum amount of

[1] *Biochem. Jour.*, 32: 10–12, 1938.