size of a crater increases with the total energy released by impact, the presence of coesite at the Wabar crater suggests that, in siliceous rocks, impact craters of this or greater size should contain coesite. The Wabar crater is comparable in size to a crater made in alluvium by the explosion of a nuclear device at a depth of about 67 feet. This device had a yield of an equivalent of 1.2 kilotons TNT (*6*).

Natural coesite has thus far been found only in materials associated with craters of presumed impact origin where shock pressures exceeding 20 kb are thought to have occurred. We shall continue to check other possible, though unlikely, sources of coesite in deformed rocks of tectonic and volcanic origin. The evidence so far, however, supports the conclusion (*1*) that coesite is a good indicator of craters formed by impact (*7*).

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7. We are indebted to Dr. Virgil Barnes for the use of his Al Hadida specimens and to Edward Henderson of the U.S. National Museum who made the specimens available to us. We also wish to thank the Arabian American Oil Company for the oblique aerial photograph of the Wabar crater.

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Active Transport of Calcium by Rat Duodenum in vivo

Abstract. In studies of the living rat, it was observed that ionic calcium is transferred against a concentration gradient and an electropotential gradient by the duodenal membrane; this would constitute evidence for the active transport of calcium in intestinal absorption and corroborates earlier observations made in vitro. The significance of this process in regard to total calcium absorption is unknown at present.

Recently, Schachter and Rosen (*1*) have postulated the existence of an active transport system for calcium by the rat intestine; they used everted duodenal sacs in vitro. The evidence that suggested active transport was primarily (i) that calcium (labeled with Ca^{45}) was transferred against a concentration gradient and (ii) that the process was inhibited by metabolic poisons. Verification of these in vitro observations was reported by Rasmussen (*2*) in studies of the effect of the parathyroid on calcium transport, by Wasserman (*3*) in studies of the metabolic basis of calcium and strontium discrimination, and by Harrison *et al.* (*4*) in studies of the effect of vitamin D on calcium absorption. It was further shown that the degree of calcium transport was correlated with the physiological need for this ion.

Table 1. Calcium fluxes across rat duodenum and a comparison of observed and theoretical flux ratios of calcium. Values for flux represent mean plus-or-minus standard error of the mean of five to six animals. Total plasma calcium was 2.6 ± 0.5 mmole, the estimated ionic plasma calcium was 1.3 mmole, and the mean potential difference between lumen and plasma was −8.1 mv.

Infused Ca (mmole)	Calcium flux (μmole/cm hr)			Ratio $\frac{Ca^{++}_{lumen}}{Ca^{++}_{plasma}}$	Flux ratio (Ca efflux/Ca influx)	
	Efflux	Influx	Net efflux		Theoretical*	Observed
0.30	0.043 ± 0.004	0.025 ± 0.003	0.018 ± 0.002	0.23	0.12	1.8 ± 0.3
0.56	.069 ± .005	.021 ± .008	.048 ± .005	.43	.23	3.2 ± .5
1.10	.11 ± .01	.034 ± .008	.076 ± .005	.85	.45	3.3 ± .9
1.97	.17 ± .02	.015 ± .004	.16 ± .01	1.52	.81	11 ± 1

* Calculated from diffusion equation of Ussing (*8*), as follows:

$$\frac{\text{(Ca efflux/Ca influx)}}{} = (C_L/C_P) \exp ZF(\psi_L - \psi_P)/RT,$$

where C_L and C_P = concentration of ionic Ca in lumen and plasma, respectively; Z = ionic charge; R = gas constant; F = Faraday; T = absolute temperature; and $\psi_L - \psi_P$ = potential difference between lumen and plasma.

Since the in vitro biological preparation is certainly nonphysiological, the argument has been put forth that observations thereon cannot be taken to mean that the active transport of calcium occurs in the intact animal. Also, in the above in vitro studies, the potential differences across the membrane were not measured. It was important, therefore, to examine in detail the kinetics of calcium transfer across the intestine of the living animal and to evaluate transport by accepted, classical procedures. The present study (*5*) was undertaken for this purpose; it was based upon the technique of Curran and Solomon (*6*).

Male albino rats (Carworth) weighing 200 to 250 g were fasted overnight before use. Surgical anesthesia was produced with sodium pentobarbital (about 6 mg/100 g body weight); the animal was maintained in this state by periodic injections of the drug. After laparotomy, an incision was made in the gastric wall, and polyethylene tubing was inserted through the stomach into the upper duodenum; the tubing was tied in place with suture. The outflow cannula and a saturated potassium chloride agar bridge were inserted into the duodenal lumen at a distance of 10 to 15 cm from the pylorus; they were then tied in place. The other agar bridge was placed in the peritoneal cavity adjacent to the duodenum. The agar bridges were led into calomel electrodes which, in turn, were attached to a sensitive electropotentiometer. The potential across the membrane was measured both directly and by determining the voltage necessary to null the measured potential. Measurements were made of the potential gradient between blood (carotid arterial and jugular venous blood), peritoneal cavity, and lumen. It was observed that the potential gradient between lumen and blood and between lumen and peritoneal cavity differed only by about 1 mv; therefore, the lumen-peritoneal potential closely approximated that of the lumen-plasma potential.

To test further the reliability of the measurement, the potential was determined when both electrodes were placed within the peritoneal cavity; here, the observed potential gradient ranged between +2 and −2 mv, indicating errors due to placement differences and differences in electrodes. Further, it was found that the potential dropped to zero when the animal was killed with one electrode in the lumen and the other electrode in either the blood stream or in the peritoneal cavity. Thus, any observed potential greater than +2 mv or less than −2 mv during the infusion studies was taken as an indication of a true potential across the membrane.

The intestine was left exteriorized and kept moistened by covering with saline-saturated absorbent cotton. The animal with attached electrodes and tubing was placed within an incubator maintained at 37°C.

The infusion solutions were made by dissolving the appropriate amount of calcium chloride (Mallinckrodt) in sterile, nonpyrogenic physiological saline. Radioactive calcium (Ca^{45}) was added at levels of 0.5 to 2.0 μc/ml. The fluids were infused in order of increasing strength at controlled rates, usually 0.0388 ml/min, by the use of a motorized syringe pump (Harvard Apparatus Co.). Before collections were made, sufficient fluid was passed through the intestine to assure that the previous solution had been completely

replaced. The solution flowing from the distal cannula was collected in graduated tubes for determining volume outflow per unit time. The rate of water absorption was determined by the difference between inflow and outflow rates of the solutions. Aliquots of the incoming and outgoing fluids were analyzed for calcium, sodium, and potassium by flame photometry; the radionuclide was estimated by standard radioisotopic procedures with a thin-window Geiger-Muller counter. At termination, the rats were bled by cardiac puncture. The plasma was collected and analyzed for total calcium, total protein by the biuret method, and radiocalcium.

Efflux is defined as the unidirectional movement of ion from lumen to plasma, and influx as the unidirectional movement from plasma to lumen. Net calcium flux is the difference between efflux and influx rates. The various flux rates, in micromoles per centimeter per hour, were calculated from the following equations:

$$\text{Ca efflux} = \frac{(Ca^{45}{}_i)(W_i) - (Ca^{45}{}_o)(W_o)}{[(SA_i + SA_o)/2]\,(L)} \quad (1)$$

$$\text{Net Ca flux} = \frac{(Ca^{40}{}_i)(W_i) - (Ca^{40}{}_o)(W_o)}{L} \quad (2)$$

$$\text{Ca influx} = (\text{Ca efflux}) - (\text{net Ca flux}) \quad (3)$$

The symbols are defined as follows: Ca^{45} = radiocalcium content of fluids in counts per minute per milliliter; Ca^{40} = total calcium of fluids in micromoles per milliliter; SA = specific activity of calcium in fluids in counts per minute per micromole; W = rate of water flow in milliliters per hour; and L = length of duodenal segment in centimeters. The subscripts i and o indicate inflowing and outflowing solutions, respectively. The assumptions in these calculations are that the backflow of Ca^{45} from plasma to lumen is small, and that the mean specific activity of calcium in the intestine is approximated by the average of the specific activities in the inflowing and outflowing fluids. Since the incoming and outflowing specific activities differed by only 20 percent, it was estimated that the error in using the arithmetic mean instead of the geometric mean would be small.

The essential data on the unidirectional fluxes and net transfer of calcium across the rat duodenum are presented in Table 1. First, it may be noted that the efflux and net flux of calcium increased with increasing concentration of calcium ion in the lumen; however, the influx rates were not significantly altered although there may have been a depression at the highest level of calcium (1.97 mmole). Especial note should be given to the observation that there was a net efflux of calcium at even the lowest calcium concentration. The net flux of water also was unaffected by calcium concentration or the length of time that the animal was under study. Although the data are not included here, it was further observed that the flux rates of calcium were not correlated with the net movement of sodium from lumen to plasma or with the net movement of potassium from plasma to lumen.

Total plasma calcium was about 2.6 ± 0.5 mmole (mean ± standard error of the mean) and within the normal range for the rat. The ionic calcium in plasma, as estimated from the nomograph of McLean and Urist (*7*) relating total plasma calcium, total plasma protein, and ionic plasma calcium, was found to be about 1.3 mmole, or 50 percent of the total. The potential gradient from lumen to plasma averaged about −8.1 mv; with these values and the concentration of ionic calcium in the lumen, the theoretical flux ratio was calculated from the diffusion equation of Ussing (*8*), as given in the footnote of Table 1. If the transfer of calcium from lumen to plasma and from plasma to lumen was due only to passive physicochemical forces, the observed ratio would be approximately the same as the theoretical ratio. From Table 1, it may be seen, however, that the observed flux ratio exceeded the theoretical ratio at all levels of calcium concentration in the inflowing solution. According to the usual definitions, this would constitute evidence for the active transport of calcium in the intact animal and, therefore, substantiates observations made in vitro. The contribution of this process to the total absorption of calcium by the intestine, and its importance in calcium homeostasis, is unknown at present and remains to be assessed.

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Olfactory Bulb Response of Rabbit

Abstract. An approach to understanding the properties of dendrites is to record the response of the olfactory bulb where the dendrites of mitral cells form the glomeruli. After the stimulations of the bulb and nasal mucosa, the responses appear different, but they are fundamentally composed of three successive potentials, suggesting that the last one is the action potential of glomerular dendrites.

It has been suggested that brain waves are due to the synchronization of dendritic potentials lasting 10 to 20 msec (*1*). Therefore, we studied the direct cortical response of the olfactory bulb because of its relatively simple cortical lamination. In a preliminary report (*2*) we showed that the bulb response induced by direct stimulation is composed of two successive potentials, referred to as the spike and slow potential. It seems probable that the spike potential is assigned to an action potential in olfactory nerves, the slow potential to the activity of glomerular dendrites.

Recently, Ottoson (*3*) reported that the olfactory bulb response, lasting about 150 msec, is induced by electrical stimulation of the nasal mucosa of frog and is composed of two waves, P_1 and P_2. It is thought that P_1 is a synaptic potential arising in the glomeruli, while P_2 is due to the propagated activity in secondary neurons. The present report is confined to the analysis of the potential form that can be induced by direct stimulation of the olfactory bulb and nasal mucosa.

Rabbits were anesthetized with urethan (1 g/kg), and the olfactory bulb was exposed by removal of the overlying bone and dura. Stimulation (with a 0.03- to 0.1-msec pulse) was applied bipolarly to the bulb or nasal mucosa through the hole made in the ethmoid bone. Surface recording was made from a monopolar silver-silver chloride electrode with a tip 0.1 mm in diameter. The temperature around the subject was kept at 28° to 30°C with a heating device. All recordings were made with a resistance-capacitance coupled amplifier of time constant 0.03 second. Figure 1 (*A* and *B*) demonstrates wave forms of the response recorded 1 to 2 mm from the site of stimulation, and the graded nature with increasing stimulus strength.

When it is fully developed, the response is composed of two potentials, a spike and a succeeding slow potential. The amplitude of the spike potential increases almost linearly without any detectable steps when the stimulus strength is increased. However, the slow