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Impact of Neodecortication on Colon Motor Response to a Meal in the Rat

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The colon motor response to a meal consisting of 100 mM of sodium oleate was assessed before and after neodecortication in male Sprague-Dawley rats. Recording probes were anchored surgically in the ascending and descending colon. Pressure changes were recorded on a dynograph using a low-compliance perfusion system. A motility index took into account the amplitude, duration, and frequency of contractions. Neodecortication increased the motility index of the distal colon in the fasting state. However, removal of the cerebral cortex did not affect significantly the colon motor response to a meal. Meal stimulation increased the motility index before and after neodecortication. These findings suggest that (1) resting colonic motor activity is increased after neodecortication, probably through the loss of an inhibitory influence of the central nervous system; and (2) the cerebral cortex is not required for the colon response to a meal in the rat.

KEY WORDS: neodecortication; mortor activity; colon response to a meal.

Eating has been noted to increase the motor activity of both the proximal and distal colon (1). A colon response to eating has been observed during consumption of the meal by some (2), early and late after ingestion by others (3), or not at all by yet other investigators (4).

The central nervous system modulates many gastrointestinal functions. Emotional stress can stimulate colon motor activity in man (5, 6). In the cat, stimulation of cerebrum, midbrain, and hypothalamus increased the colon motor activity (7). The role of the central nervous system on the colonic response to a meal is, however, poorly understood. This kind of response may be modulated at different levels of the central nervous system; the sight and smell of the meal through the cerebral cortex, gastric distension through midbrain, gastric emptying and proximal intestinal distension through the spinal cord.

The purpose of the present study was to evaluate the impact of a telencephalic structure of the central nervous system, the cerebral cortex, on both proximal and distal colon motor response to eating.

MATERIALS AND METHODS

Male Sprague-Dawley albino rats weighing 296 ± 47 (mean ± SD) g were used. The animals had free access to water and Purina Rat Chow (Check-R-Board, Novi, Michigan). Each animal was anesthetized with an intraperitoneal injection of 250 mg/kg chloral hydrate, the abdomen opened by a midline incision, and the colon was exposed. The recording probe was a 20-cm segment of polyethylene tubing (PE-90, Clay-Adams Intromedic, Parsippany, New Jersey) with an internal diameter of 0.86 mm and a side opening of 0.5 mm located 1.0 cm from the

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end of the catheter. Recording probes were introduced into the proximal and distal colon through a 2-mm incision. They were secured with encircling ligatures, while maintaining the side openings oriented toward the colonic lumen. The proximal recording site was therefore 2–3 cm distal to the cecum, and the distal one was about 5–6 cm proximal to the anus. Care was taken not to damage the parallel vasculature and to maintain the organs in their *in situ* positions. The recording probes were led under the skin and brought out at the back of the neck where a 4-cm length of the tubing was left exposed. The abdominal wall was closed and the animal returned to its home cage to recover.

A baseline recording was performed at least 48 hr after the pressure probes had been implanted. Following a 24-hr fast, a resting motor activity was recorded over 30 min. Each animal then received a meal consisting of 100 mM sodium oleate. The meal was introduced into the stomach by a syringe attached to a cannula. A piece of soft rubber tubing was fixed on the tip of the cannula to protect the esophagus against trauma. Recordings continued for 90 min after the meal was administered.

Each pressure probe was perfused at the rate of 0.5 ml of water per minute by a low-compliance pneumohydraulic capillary system (Arndorfer Medical Specialties, Greendale, wisconsin). Intraluminal pressure changes were recorded by a volume displacement transducer (type 4327-I, Bell and Howell, Pasadena, California) and a rectilinear dynograph (Beckman Instruments, Fullerton, California) which was calibrated before each study. All records were coded and analyzed blindly by one observer (D.H.) to minimize errors that might result from variations in interpretation of the contractions or artifacts. The motor activity of the colon was quantified by determining the number of waves and their amplitude and duration. A motility index (MI) was calculated as follows (8): motility index = $n(AD)/(T10)$ in which A = amplitude, D = duration, T = recorded time in minutes, and n = number of waves during T. To avoid artifactual errors, waves of less than 3 mm Hg were excluded from analysis (9). Each peak of a complex wave was counted as one wave.

Following a baseline recording, the animals were subjected to cerebral neodecortication. The scalp was incised in midline and retracted. Holes were drilled on each side of the skull and widened to expose most of the frontal, parietal, temporal, and occipital cortices. The dura was reflected, and the cortical tissue was removed by aspiration. The skin was then closed with clips after bleeding had stopped. The effect of the sodium oleate meal on the colon motor activity was repeated at least seven days after cerebral neodecortication.

The mean motility indices of each postneodecortication period were compared with those of the preoperative period. Since the data points were not normally distributed, the data were also analyzed by a nonparametric method such as Wilcoxon's rank-sum test. Postmortem examination was performed to confirm the location of the recording probes in the colon and evaluate the extent of neodecortication.

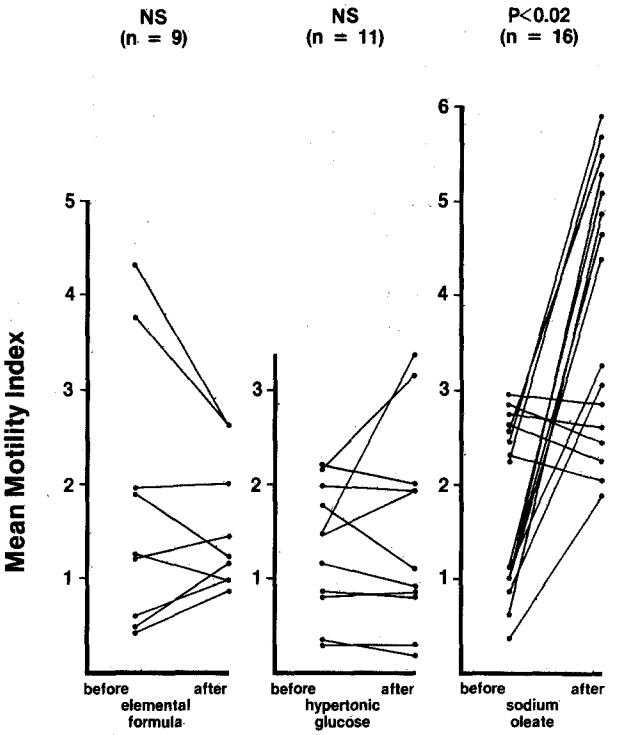

Fig 1. Comparison of the colon motor response to sodium oleate with that of hypertonic glucose and an elemental diet formula. The values for 30 min before and after the meals are compared. Only the sodium oleate meal significantly increased the motility index.

RESULTS

In a preliminary study, the colon motor response to 2 ml of an elemental diet formula (Ensure-Plus, Ross Laboratories, Columbus, Ohio), 20% glucose, or the same volume (100 mM) of sodium oleate was evaluated. The values for 30 min before and after the meals were compared. The motility indices (mean ± SD) before and after the elemental formula were 1.80 ± 1.39 and 1.61 ± 0.72. These values for glucose were 1.11 ± 0.70 vs 1.59 ± 1.02 and for sodium oleate 1.75 ± 0.87 vs 3.89 ± 1.42, respectively. The response to sodium oleate was more impressive and consistent (Figure 1). It was only the sodium oleate meal that significantly increased the mean motility index ($P < 0.02$). Increased motor response was noted in 69% of the experiments.

The effect of sodium oleate meal on the distal colon motor activity of the rat is shown in Figure 2. A cluster of contractions usually was noted within 30 min from administration of the meal. The frequency of contractions markedly increased after the meal. Both the number and amplitude were generally higher in the distal than proximal colon. The

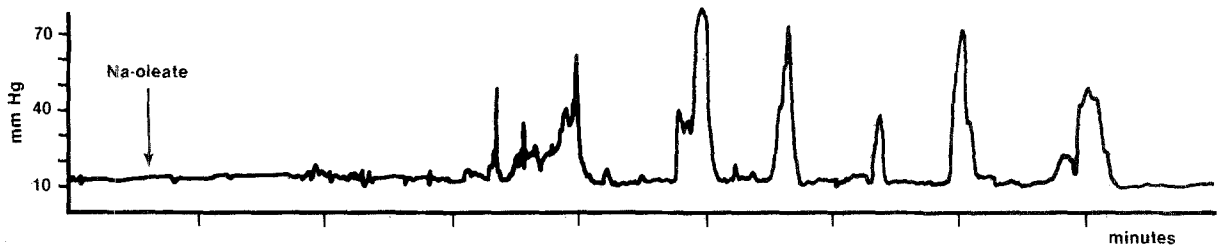

Fig 2. The effect of sodium oleate meal on the distal colon motor activity. A cluster of contractions usually was noted within 30 min from administration of the meal.

elemental diet formula and hypertonic glucose did not increase the mean motility index. Therefore, sodium oleate was utilized in the rest of the experiments.

A group of nine animals underwent cerebral neodecortication. The mean motility index of the distal colon for each 30-min period before and after neodecortication is shown in Figure 3. In the fasting state, the motility index was significantly greater after neodecortication: 0.82 ± 0.40 vs 1.78 ± 0.51 (mean ± SD) $P < 0.001$. Sodium oleate significantly ($P < 0.01$) increased the motility index before and after neodecortication. Removal of the cerebral cortex did not affect significantly the colonic motor response to the meal.

In the proximal colon the response to the meal was not as clear as in the distal colon; therefore, the impact of the cerebral neodecortication could not be evaluated in a meaningful way.

DISCUSSION

Food ingestion is said to be the major physiologic stimulus to the motor activity of the colon. Fat usually produces a prolonged and stronger response in the colon than a heterogeneous meal (10). In the present study, we made a similar observation when we compared the colonic response to sodium oleate with that occurring after elemental formula. The mechanism of such a colonic motor response, however, has been the subject of much controversy. It is understood that the response is independent of food reaching the large bowel (11). A complex interaction between afferent receptors in the gastroduodenal area and cholinergic and opioid neurons (12) in the enteric nervous system seems to play a major role in this process.

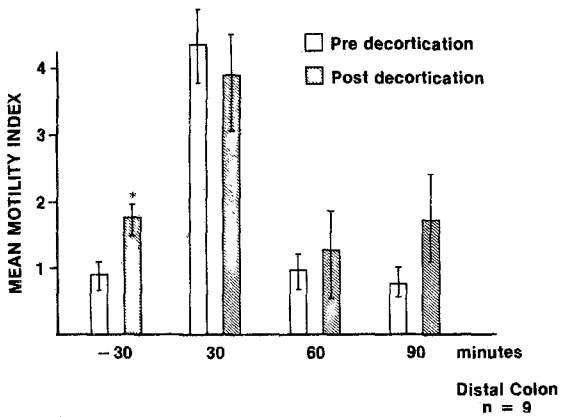

Fig 3. Sodium oleate increased the motility index of the distal colon before and after neodecortication. In the fasting state, however, neodecortication significantly increased the motility index. Values are mean ± SD.

Despite numerous studies (7, 13, 14) concerning the effect of the cerebral cortex on basal colonic motor activity, little is known about its influence on the colonic response to a meal. The data reported here clearly indicate that neodecortication increases the basal colon motor activity while it does not influence the colon motor response to a meal. In a previous study (15), the resting colon motor activity was only transiently affected following spinal cord transection. By the end of the first week, the resting motor activity gradually recovered to the preoperative level. On the basis of this observation, we elected to repeat the experiments at least seven days after neodecortication surgery.

The increase in basal motor activity suggests the loss of an inhibitory influence from cerebral cortex on the distal colon. This is consistent with Rostad (7) who elicited the inhibitory role for several segments of the cortex on colon motor activity. These findings are further in accord with those of Sun and her colleagues (12) who found that sham feeding did not initiate a colon motor response. These observations suggest that probably there is no cephalic phase to the colon response to a meal.

We conclude that (1) feeding a fat meal to the rat increases the motor activity in the colon; (2) this response is more prominent in the distal colon; (3) resting colon motor activity is increased after

neodecortication; this may be through the loss of an inhibitory influence of the central nervous system; and finally (4) the colon motor response to a meal is not abolished by neodecortication.

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