GABA[8]. The difference between our depth recordings of unchanged polarity of the slow negative wave and those of Pollen and Sie[7] (they found positive counterpart in the 5th cortical layer) must be solved experimentally.
Our results allow us to draw the conclusion that the nature of 2 negativities, the spike and the wave components of cortical focal discharges, is different.

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Naloxone and intestinal motility

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Summary. It was supposed that the inhibition of intestinal peristalsis seen in animals and humans after abdominal surgery might be related to the release of endorphins, endogenous opiate receptor agonists, caused by the surgical stress and pain. However, naloxone, a potent morphine and endorphin antagonist, failed to block this peristaltic inhibition in rats, which leaves the mechanism of this inhibition, and thus the function of intestinal endorphins, still very much in doubt.

There is evidence that the brain contains endogenous substances that act as agonists at morphine receptor sites[1-4]. These substances, called endorphins, are localized in specific neurons, especially along the pain pathways and the limbic system[1,3-5]. Surprisingly large quantities of endorphins are also found in the intestinal tract, especially in the longitudinal muscle layer and the myenteric plexus[2,6]. The function of endorphins in the intestinal tract is not known, but it has been shown that they are released by physical stress[3,5] or electrical stimulation in vitro[4,6-8]. Several investigators have proposed that their function in the intestinal tract is to regulate intestinal motility[8-11]. In vitro, exogenous endorphins relax or inhibit contractions of isolated intestinal segments. These and most other effects of endorphins, both in vitro and in vivo, can be blocked by the morphine antagonist naloxone[4,5,7-11]. The present study was initiated to investigate the effect of naloxone on intestinal motility in surgically stressed, conscious animals in order to determine whether the inhibition of intestinal motility seen after surgical stress might be due to release of endogenous endorphins.
Methods. Male Wistar rats (Simonsen Laboratories) were kept in a 12:12 light-dark cycle (lights on at 6.30 h). All experimental procedures began at 9.00 h. Rats to be subjected to surgical stress were anesthetized with ether, their abdomens were opened, their cecums were ligated and removed, and the wound was closed. 2 h after surgery, animals received, orally, 2 ml of a 10% charcoal meal (10% carbon black in 1% hydroxypropylmethylcellulose). 45 min later, they were killed, their small intestines were carefully removed, and the progression of the charcoal meal down the intestine was measured. This was expressed as a ratio to the total length of the small intestine, or 'percent transit'.
Control animals also received 2 ml of the charcoal suspension and were killed 45 min afterward. Naloxone in saline was administered s.c., either 5 mg/kg at the time of surgery and 2 mg/kg 20 min before the charcoal, or 0.1 or 2 mg/kg 20 min before the charcoal administration. Controls were given saline alone, at these times.
Results. The stress caused by anesthesia and cecectomy had a very dramatic effect on gastrointestinal transit of the charcoal, as shown in table 1. The s.c. administration of naloxone (2 mg/kg to unoperated animals) 20 min before the charcoal had no effect on the gastrointestinal charcoal transport.
When naloxone was administered to operated animals, no reversal of the stress-induced depression in gastrointestinal transport of charcoal occurred over a wide range of naloxone doses (0.1 mg/kg to a total of 7 mg/kg). The slight opposite tendency toward enhanced depression becomes significant ($p<0.05$) when all experiments are combined (table 2).
Discussion. No effects of naloxone on normal intestinal motility in vivo or in vitro have been observed by other workers[3,12]. Similarly, no effects were found in our experiments despite the fact that several investigators have reported a release of endorphins from the myenteric plexus under stress[5,7,9,10]. Endorphins released in this manner could be expected to inhibit intestinal motility. The administration of naloxone in these cases should then block the action of endorphins and increase intestinal motility. In guinea-pig ileum in vitro, when peristalsis is elicited by

Table 1. Effect of cecectomy or naloxone on gastrointestinal transit of a charcoal meal

Treatment	N	Transit (%)	p
Nonoperated controls	8	75.1±2.1	-
Nonoperated and naloxone (2 mg/kg)	5	75.0±3.9	NS
Operated controls	14	20.9±1.5	<0.01

Table 2. Effect of naloxone after cecectomy on gastrointestinal transit of a charcoal meal

Experiment No.	Operated controls		Naloxone treatment			p
	N	Transit (%)	N	Dose	Transit (%)	
1	4	21.3±2.7	4	2 mg/kg	15.4±0.9	<0.1
2	4	19.4±3.7	4	2 mg/kg	17.1±1.2	>0.1
			5	2 mg/kg+ 5 mg/kg	17.4±1.9	>0.1
3	6	21.7±2.2	6	0.1 mg/kg	18.9±1.7	>0.1
Overall	14	20.9±1.5	19		17.4±0.8	<0.05

distension, the peristalsis slowly fatigues; this effect can be reversed by naloxone[10].

Previous experiments have shown that the inhibition of propulsive intestinal motility that occurs following this type of abdominal surgery in rats mimics the postoperative paralytic ileus seen in humans[13]. This inhibition is not clearly related to release of known neural transmitters in the intestine. Therefore, it seemed possible that endorphins could be involved and that administration of naloxone would block - and thus reveal - this influence. In our experiments, however, no such response to naloxone occurred, which implies that the decreased intestinal motility following cecectomy is not related to release of endogenous endorphins in the intestine (or from the brain, as a central response to pain). The paralytic ileus seen in humans after intestinal surgery may thus be similarly unrelated to release of endorphins. It is unclear whether the slight decrease in propulsive intestinal motility following naloxone administration to operated animals has anything to do with endorphins.

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A rapid method for measuring the phospholipid synthetic activity of incubated lymphocytes

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Summary. A rapid and simple method for estimating the phospholipid synthetic activity of incubated lymphocytes by a Florisil column technique following the uptake of ^{14}C-oleic acid was established. Stimulation of phospholipid synthesis by PHA and inhibitions caused by Tween 20 or Tween 80 and heating were evaluated easily with this method.

It is known that the phospholipids are synthetized rapidly in cultured lymphocytes following the administration of various precursors such as fatty acids, phosphate etc. Incorporation of ^{14}C-oleic acid, ^{14}C-acetate[1], ^{32}P-phosphate[2], ^{14}C-glycerol and ^{14}C-choline[3] into phospholipids extracted from lymphocytes is enhanced after the stimulation of mitogens such as PHA, ConA etc. On the other hand, these incorporations are supposed to be inhibited following impairment of lymphocytes.

We developed a rapid and simple method for estimating the phospholipid synthetic activity of lymphocytes by measuring labelled phospholipids, which were extracted from the incubated cells and purified by Florisil column.

Lymphocytes were separated from lymph nodes of male Wistar rats weighing 150-220 g following the method reported previously[4]. Separated lymphocytes were suspended in RPMI-1640 solution (GIBCO, Grand Island, USA) to be 2×10^7 cells/ml. Usually, ^{14}C-oleic acid was applied as a

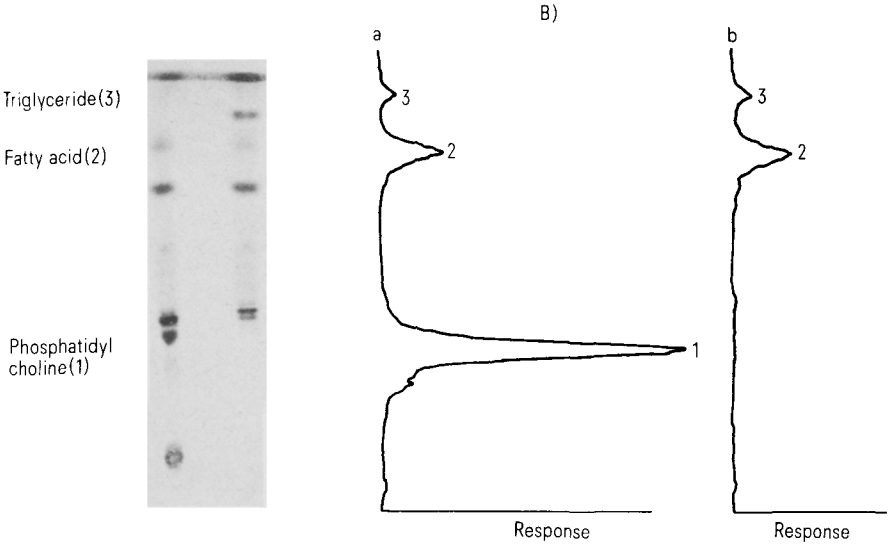

Fig. 1. Thin layer chromatograms (A) and radiochromatograms (B) of the lipids extracted from the incubated lymphocytes before and after the Florisil column chromatography. *a* Before the column treatment; *b* after the treatment (effluent). In both chromatograms A and B, phosphatidyl choline indicated as 1 in *a* disappeared completely.