STIMULATION OF THE MECHANORECEPTORS OF THE INTESTINAL WALL AS A CAUSE OF DYNAMIC INTESTINAL OBSTRUCTION

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(Presented by Academician V. N. Chernigovskii)
Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 55, No. 5, pp. 45-48, May, 1963
Original article submitted June 8, 1962

Distension of loops of bowel invariably accompanies diseases such as acute intestinal obstruction and peritonitis. The significance of this factor to the mechanism of death from intestinal obstruction has been studied by several workers. It has been found that distension of an isolated Thiry-Vella loop of intestine leads to the development of a pathological state clinically resembling acute intestinal obstruction, and leading to death of animals within 7-8 days from the beginning of distension [5, 6]. It has also been demonstrated that preliminary denervation of the loop or division of the vagus nerves and removal of the abdominal sympathetic ganglia completely abolishes the effects of prolonged distension. These findings show that the developing process is reflex in nautre. Several aspects of the mechanism responsible for the disturbances arising from distension of the bowel loop require further study; in particular, we have no information concerning the character of the changes in the motor activity of the small intestine.

Other researchers [1, 3, 4] have shown that stimulation of the mechanoreceptors of the alimentary canal causes marked inhibition of the motor function of the small intestine. Moreover, during intensive distension of any segment of the intestine inhibition of its motor activity may be recorded along the whole length of the small intestine [2]. Reflex inhibition of the motor function of the small intestine has been studied only by P. G. Bogach [1], who showed that intensive stimulation of the mechanoreceptors of the rectum may lead to prlonged (up to 24 h) inhibition of the motor activity of the alimentary tract.

The object of the present investigation was to study the disturbance of the motor activity of the small intestine during prolonged stimulation of the mechanoreceptors of the intestinal wall, to investigate the mechanisms underlying this reaction, and to study the role of the motor disturbances in the development of the pathological process developing during distension of a loop of bowel.

EXPERIMENTAL METHOD

Experiments were carried out on 11 dogs with a Thiry-Vella or Thiry-Herrin loop. The Thiry-Herrin operation differs from the other by the fact that the caudal end of the loop is not brought out under the skin, but is anastomosed end-to-side to part of the small intestine. The areas of intestine for formation of the Thiry-Vella and Thiry-Herrin loops were excised from different portions of the jejunum and ileum in the different experiments. In some experiments the Thiry-Herrin loop was sutured to the jejunum, in others to the ileum. An isolated Thiry-Vella loop of intestine was distended by introducing a rubber balloon, 10-15 ml in volume, the pressure inside which was measured by a mercury manometer. Changes in the motor activity of the small intestine were recorded by means of a rubber balloon, 0.8-1.5 ml in volume, introduced on a catheter into the lumen of the bowel through the Thiry-Herrin loop. The catheter was connected through a water manometer to a V. F. Mostun's (a modification of Marey's) capsule. The motor activity of the intestine was recorded in ink on the drum of a kymograph.

EXPERIMENTAL RESULTS

Inflation of the balloon in the intestinal loop to 60-80 mm Hg caused, as a rule, complete inhibition of the "fasting" motor activity, regardless of which portion of the intestine was isolated as a Thiry-Vella loop and of the part of the intestine in which the motor activity was recorded. The "satiated" motor activity was inhibited when the

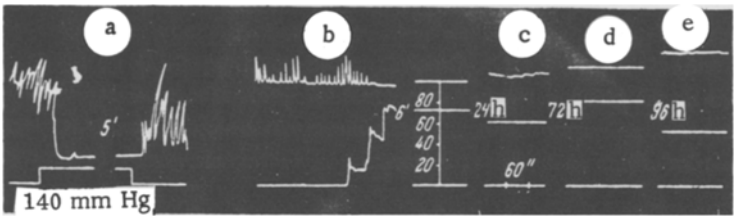

Fig. 1. Inhibition of motor activity of the small intestine during brief (a) and prolonged (b, c, d, e) distension of the Thiry-Vella loop of bowel. Significance of the curves (from above down): balloonographic recording of the contractions of the small intestine; pressure inside the balloon distending the Thiry-Vella loop; zero pressure line.

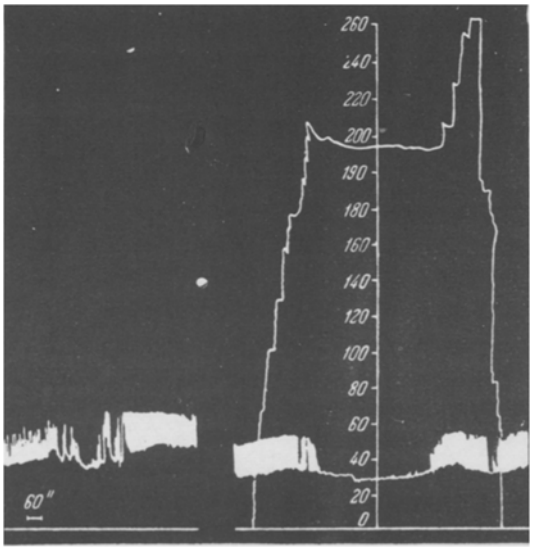

Fig. 2. Effect of stimulation of the mechanoreceptors of the Thiry-Vella loop of bowel on the motor activity of the small intestine of an anesthetized animal. Significance of the curves (from above down): pressure inside the balloon; recording of contractions of the circular muscle of the intestinal wall; zero pressure line.

balloon was inflated to 100-160 mm Hg (Fig. 1, a). Inhibition of the motor activity lasted even when the mechanoreceptors of the intestinal wall were stimulated, and dynamic intestinal obstruction developed. Immediately after the balloon began to be inflated, the animals in some experiments vomited and in most experiments they developed motor excitation, which lasted for the first 2-3 h of the experiment. Release of air from the balloon at this stage of the experiment restored the animal's condition and motor activity to normal within 10-40 min. If the mechanoreceptors were stimulated for a longer period (up to 48 h), generalized excitation gave way to a state of depression, periodic vomiting, and loss of appetite. If distension was discontinued at this stage the animal's condition again returned to normal, but the dynamic intestinal obstruction took longer to be relieved: motor activity was only restored after 2-6 h. In two experiments in which distension of the loop of bowel was discontinued after 4 and 5 days the motor activity did not recover (Fig. 1, b, c, d, e) and the animals died with signs of increasing paralytic intestinal obstruction on the 6th and 8th days of the experiment.

To explain the mechanisms of reflex inhibition of motor activity during stimulation of the mechanoreceptors of the intestinal wall we repeated the same experiment in animals anesthetized with morphine and thiopental or receiving injections of the ganglion-blocking agent dicoline in doses of 0.5-1 mg/kg.

After morphine-thiopental anesthesia, distension of the loop of bowel caused inhibition of the motor activity of the small intestine, but for a short time only; the activity was restored even though distension continued. An increase in the pressure inside the balloon was followed by an even shorter period of inhibition of motor activity. Consequently, even prolonged distension under a pressure of 200-260 mm Hg had no inhibiting effect on the motor activity of the small intestine (Fig. 2). Morphine-thiopental anesthesia, if induced while motor activity was actually inhibited as a result of stimulation of the mechanoreceptors of the intestinal wall, led to restoration of the motor activity of the small intestine.

Stimulation of the mechanoreceptors of the Thiry-Vella loop after administration of the ganglioplegic did not produce inhibition of the motor activity when the stimulation was of the usual strength (60-80 mm Hg), but inhibition was obtained by much stronger stimulation (about 200 mm Hg). The inhibition thus produced was just as persistent as in the original experiments. Administration of dicoline during inhibition of motor activity caused by distension of the loop to 80-100 mm Hg led to its restoration. If the pressure inside the balloon was raised still further the motor activity was inhibited, and this inhibition lasted as long as the high pressure continued; a lowering of the pressure inside the balloon to 100 mm Hg again led to restoration of the motor activity (Fig. 3).

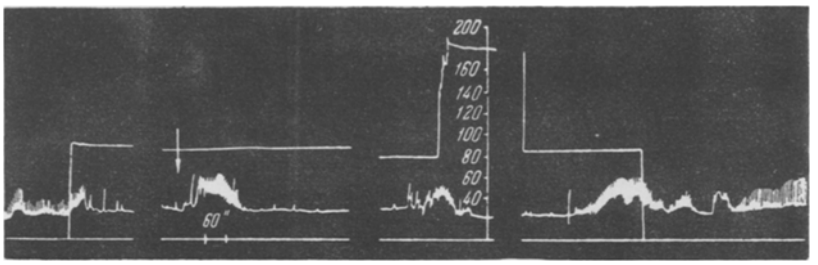

Fig. 3. Effect of distension of the Thiry-Vella loop of bowel on the motor activity of the small intestine after administration of the ganglioplegic drug dicoline. Significance of the curves (from above down): pressure inside the distending balloon; tracing of contractions of the circular muscle of the intestine; zero line of pressure inside the balloon. The arrow marks the time of injection of dicoline.

Stimulation of the mechanoreceptors of the intestinal wall, whatever its intensity, following morphine-thiopental anesthesia or injection of dicoline, did not cause inhibition of the motor activity of the small intestine.

Injection of dicoline in the course of prolonged stimulation of the mechanoreceptors relieved the paralytic intestinal obstruction, even on the 5th day after the distension began, i.e., when the discontinuation of the distension itself did not prevent death of the animal. Immediately after restoration of the motor activity the vomiting ceased, appetite reappeared, and the characteristic adynamia disappeared within a short time.

These experiments demonstrated that prolonged stimulation of the mechanoreceptors of the intestinal wall leads to the development of dynamic (paralytic) intestinal obstruction, revealed not only by total inhibition of motor activity, but also by a characteristic clinical picture: vomiting, anorexia, and depression. It is evident that dynamic intestinal obstruction is an important link in the development of the pathological process during prolonged distenstion of the intestinal loop.

We may conclude from these results that reflex inhibition of the motor activity of the small intestine is brought about in two ways. The first is mediated through the autonomic ganglia, and may produce a transient inhibition of the motor activity. Ganglion block interrupts this reflex path. The reflex arc of the second inhibitory reflex, giving rise to prolonged inhibition of the motor activity, is not interrupted in the ganglia, for a lasting inhibitory reaction may be obtained by strong stimulation of the mechanoreceptors after administration of ganglioplegics.

The fact that prolonged inhibition of the motor activity is abolished by general anesthesia suggests that this process is based on a true reflex, the arc of which is joined in the higher divisions of the central nervous system.

SUMMARY

As demonstrated in chronic experiments on dogs, prolonged irritation of mechanoceptors of the intestinal wall caused development of dynamic intestinal obstruction. Inhibition of the motor function is accompanied by a characteristic triad – vomiting, anorexia and depression.

Motor inhibition is caused by two different reflexes. The arc of one of them is blocked in the sympathetic ganglia and may be interrupted by administration of a gangliolytic (dicoline). The second route of reflex inhibition evidently passes through the higher portions of the central nervous system, since the morphine-thiopenthal anesthesia interrupts the arc of this reflex.

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