Electromyographic Observation of Intestinal Movement during Intestinal Obstruction*

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Narrowing of the intestinal lumen and a circulatory disturbance are the two basic factors involved in intestinal obstruction. This paper analyzes the effect of each of these factors upon the motility of the canine intestine by means of electromyography during experimentally induced mechanical obstruction and vascular occlusion. EMG in simple mechanical obstruction is characterized by an increase followed by a decrease in the intensity of spike discharges, in which course there is a period where a group of intensified spike bursts and a resting interval appear alternately. The change in EMG on vascular occlusion also consists of intensification and subsequent debilitation of the discharges, but on this occasion the progress is quite rapid and the resting interval is never observed. EMG on strangulating obstruction includes the manifestation of mechanical obstruction and that of vascular occlusion, and the former is prevalent in the case of mild strangulation and the latter in the case of acute, complete strangulation. The ease with which the intestine resumes its motility on removal of the cause of intestinal obstruction largely depends on the duration of obstruction, the maximum duration assuring a complete recovery being approximately 24 hours for simple mechanical obstruction and about two hours for vascular occlusion.

Recently, electromyogram (to be abbreviated hereafter as EMG) of the intestine has become a useful tool not only for fundamental researches but also for elucidation of clinical problems. Motor function of the intestine during intestinal obstruction, for example, has been studied so far mainly by simple observation at laparotomy, kymography, abdominal window method and x-ray-cinematography, but the electromyographic study is becoming more and more popular in this field as seen in reports by Takita et al.1) and by Nishijima2).

Needless to say, narrowing of the intestinal lumen and circulatory disturbance in the intestinal wall are the two basic factors participating in the development...
of intestinal obstruction. The significance of the latter factor may be easily understood from the anatomical fact that the intestine is provided with end arteries. However, the analytical observation of these two factors has not yet been accomplished even in the detailed study performed by Takita et al. With such a consideration, the author investigated electromyographically the motor function of the canine intestine during and after simple mechanical obstruction of its lumen, during and after occlusion of the mesenteric vessels, and during strangulation of the intestine.

MATERIALS AND METHODS

Animals: A total of 54 adult dogs were used. The animal was anesthetized with 10 to 20 mg/kg body weight of Thiopental sodium administered intravenously, laparotomized by a midline incision and subjected to electromyography of the intestine before and after the preparations described below.

Procedures for the animal intestine: Mechanical obstruction was induced by simple ligation of the ileum that involved the intestinal canal but not its mesentery, whereas circulatory disturbance was caused by clamping the superior mesenteric vessels with a vessel clamp. For producing strangulating obstruction, a loop of the small intestine was brought outside the laparotomy wound, the root of its mesentery was fixed by suture to the abdominal wall and then the loop was distorted 360°, so that obstruction of the lumen and a circulatory disturbance occurred simultaneously. After such preparations, the EMG was led from the intestinal segment shortly oral to the site of obstruction in the case of mechanical obstruction, from the involved intestinal segment in the case of vascular occlusion, and both from the oral and the involved segments in the case of strangulating obstruction.

Method of electromyography: The system consisting of nonpolarizable Ag-AgCl electrodes, an R.C. amplifier (time constant 0.05 sec.) and an electromagnetic oscillograph with an H-type vibrator was utilized and the action potential of the intestine was led by bipolar lead at an electrode distance of about 0.3 cm. The electrodes were inserted from the serosal surface to the muscular layer of the intestinal wall and were fixed there by suture.

RESULTS

1. EMG during mechanical obstruction

Twenty dogs were subjected to mechanical obstruction of the intestine and the action potential was led from the intestinal segment shortly oral to the site of obstruction. Table I summarizes the results and Fig. 1 illustrates EMG observed in the case of this group. The intestinal EMG, in general, consists of spike bursts, each of which appears with a single peristalsis and has one to several spike discharges. Before obstruction, the maximum amplitude of the spike bursts
TABLE I. Electromyographic Findings in Mechanical Obstruction

<table>
<thead>
<tr>
<th>Time since onset of obstruction</th>
<th>Maximum amplitude (mV)</th>
<th>Mean amplitude (mV)</th>
<th>Duration (sec.)</th>
<th>Spike number</th>
<th>Interval between bursts (sec.)</th>
<th>Resting interval (sec.)</th>
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<tbody>
<tr>
<td>Before obstruction</td>
<td></td>
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<tr>
<td>0.4-1.8 (1.61)</td>
<td>0.24-1.45 (1.35)</td>
<td>0.1-0.7 (0.61)</td>
<td>1-8</td>
<td>2.2-7.0</td>
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<tr>
<td>Early phase</td>
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<tr>
<td>1-3 hrs.</td>
<td>1.1-1.8 (1.46)</td>
<td>0.8-1.45 (1.22)</td>
<td>0.2-0.8 (0.75)</td>
<td>3-9</td>
<td>2.0-6.6 (4.4)</td>
<td></td>
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<tr>
<td>6-8 hrs.</td>
<td>1.2-2.4 (1.98)</td>
<td>1.0-2.4 (1.60)</td>
<td>0.4-1.6 (1.35)</td>
<td>3-9</td>
<td>2.0-5.4 (2.34)</td>
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<tr>
<td>Intermediate phase</td>
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<tr>
<td>8-10 hrs.</td>
<td>1.3-2.8 (1.88)</td>
<td>1.0-2.6 (1.68)</td>
<td>0.6-1.8 (1.1)</td>
<td>11-19 (14.3)</td>
<td>1.8-2.5 (2.2) (32.4)</td>
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<tr>
<td>10-12 hrs.</td>
<td>1.7-2.4 (1.96)</td>
<td>1.3-2.2 (1.64)</td>
<td>1.2-2.0 (1.36)</td>
<td>8-18 (14.4)</td>
<td>1.5-2.0 (1.86) (69)</td>
<td></td>
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<tr>
<td>Late phase</td>
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<tr>
<td>12-24 hrs.</td>
<td>0.8-1.8 (1.24)</td>
<td>0.6-1.6 (1.04)</td>
<td>0.4-0.8 (0.6)</td>
<td>6-16 (10.4)</td>
<td>3.6-5.4 (4.42) (127)</td>
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<tr>
<td>24-48 hrs.</td>
<td>0.6-0.8 (0.72)</td>
<td>0.6-0.8 (0.61)</td>
<td>0.6-0.8 (0.66)</td>
<td>3-4</td>
<td>3.8-8.0 (4.88) (72)</td>
<td></td>
</tr>
</tbody>
</table>

Note: Parentheses show mean values.

...ranged in the cases of this group from 0.4 mV to 1.8 mV, the duration of the bursts from 0.1 sec. to 0.7 sec., the number of spikes for each burst from one to eight, and the interval between consecutive bursts from 2.2 sec. to 7.0 sec.

The movement grossly observed as well as the electromyographic findings changed considerably with the lapse of time since onset of the obstruction, and the author divided the course into three phases; the early, the intermediate and the late phase. The early phase included the first eight hours after onset of obstruction and the intestinal movement in this period was characterized by hyperperistalsis with a regular period, known as "stenotic movement". The intermediate phase was approximately from 8 to 12 hours after obstruction. In this period, hyperperistalsis became more remarkable and was accompanied by the resting interval showing no peristalsis.

In the late phase which followed to the intermediate phase, distention of the intestine became marked, the peristaltic movement diminished and the resting interval became longer. At the same time, tonic contraction of long duration began to appear at various intervals and the condition was gradually transformed into intestinal paralysis. Details of the aspect of each phase are as follows:

**Early phase**: In about one to three hours, the intestinal segment oral to the site of obstruction became slightly distended and the peristaltic movement became hyperactive. In accordance with these gross findings, the EMG in this period revealed increases in the spike number, duration and amplitude of bursts and, on most occasions, a decrease of the interval between bursts. Such a
EMG in Intestinal Obstruction

Fig. 1. Changes in EMG following mechanical obstruction of the intestine.

Tendency became more prominent as time progressed until about eight hours after onset of obstruction, as indicated in Fig. 1 and Table 1, but the regularity of EMG was well preserved throughout the course of this phase.

Intermediate phase: On gross observation, distention of the obstructed intestine became more marked and an accumulation of gas and fluid was noted in its lumen. As regards the peristaltic movement, this phase was characterized by an alternate appearance of a group of powerful peristalsis and a resting interval showing scarce peristalsis. The EMG in this phase revealed, as shown in Fig. 1, grouping of strong spike bursts followed by a resting interval with very weak spike discharges. The group of the strengthened spike bursts consisted of 12 to 15 or 20
to 27 individual spike bursts, at 8 to 10 hours and 10 to 12 hours respectively, and the interval between bursts was much shorter than in the early phase. The maximum amplitude, the duration and the spike number of a single spike burst were each larger than in the early phase and became greater with lapse of time, as indicated in Table I. The resting interval began to appear about eight hours after onset of obstruction and its duration became longer as time progressed, being 20.4 to 42.0 sec. at 8 to 10 hours and 55 to 161 sec. at 10 to 12 hours.

Late phase: In this period which began about 12 hours after onset of the obstruction, the proximal intestine was further distended with gaseous and fluid contents. As to the intestinal movement, the moving phase and the resting interval were still clearly distinguished, but the movement gradually became less active and a tonic contraction of long duration began to appear at various intervals. More than 48 hours after onset of obstruction, the intestine was extremely distended and became dark violet brown in color, and the intestinal movement tended to disappear gradually. The EMG recorded at about 24 hours, as shown in Fig. 1, revealed a decreased incidence of spike bursts which were very small in amplitude. The interval between bursts and the resting interval were both much longer than in the intermediate phase. Such a debilitation of spike discharges became more remarkable as time progressed, as indicated in Table I, and the spike discharges were no longer observed after 48 hours. Meanwhile, such peculiar phenomena as an appearance of feeble discharges without gross movements and an absence of electric discharges despite the presence of a waving, tonic contraction of the intestine were noted in this phase.

2. EMG during vascular occlusion

Occlusion of the mesenteric vessels was performed in 11 dogs. Shortly after clamping of the vessels, the involved intestinal segment exhibited powerful contractions and hyperactive peristalsis. Such intensification of the intestinal movement became most remarkable 20 to 30 minutes after occlusion, and at that time small petechiae began to appear on the serosal surface of the intestine and mesentery, and hemorrhagic exudation became manifest. The involved segment became darker as time progressed and it was reddish purple after one hour. The intestinal movement gradually began to diminish in activity at one hour after occlusion of the vessels and was hardly observed at two hours.

The electromyographic findings of this group are summarized in Table II and an example is shown in Fig. 2. The evidence in EMG of hyperactivity of the intestine, i.e., increases in the amplitude, duration and spike number of spike bursts, appeared shortly after occlusion of the vessels, became more remarkable in the following 20 to 30 minutes, and then became less prominent. The interval between spike bursts, on the other hand, generally tended to increase from the beginning of the circulatory disturbance. At one or two hours after vascular
TABLE II. Electromyographic Findings in Vascular Occlusion

<table>
<thead>
<tr>
<th>Time since onset of occlusion</th>
<th>Maximum amplitude (mV)</th>
<th>Mean amplitude (mV)</th>
<th>Duration (sec.)</th>
<th>Spike number</th>
<th>Interval between bursts (sec.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before occlusion</td>
<td>0.4-0.8 (0.76)</td>
<td>0.2-0.75 (0.64)</td>
<td>0.2-0.6 (0.32)</td>
<td>1-4 (2.4)</td>
<td>2.0-2.5 (2.23)</td>
</tr>
<tr>
<td>5-20 min.</td>
<td>0.6-2.5 (1.13)</td>
<td>0.4-1.3 (0.94)</td>
<td>0.4-1.3 (0.86)</td>
<td>1-8 (5.3)</td>
<td>2.0-3.0 (2.69)</td>
</tr>
<tr>
<td>20-30 min.</td>
<td>2.0-3.8 (2.95)</td>
<td>1.8-3.0 (2.48)</td>
<td>0.3-0.8 (0.59)</td>
<td>3-9 (5.6)</td>
<td>2.1-3.8 (2.5)</td>
</tr>
<tr>
<td>30-60 min.</td>
<td>1.1-3.8 (2.75)</td>
<td>1.0-2.8 (2.31)</td>
<td>0.7-1.6 (0.99)</td>
<td>3-10 (7.5)</td>
<td>2.6-4.8 (3.99)</td>
</tr>
<tr>
<td>60-120 min.</td>
<td>0.1-0.6 (0.43)</td>
<td>0.1-0.4 (0.33)</td>
<td>0.1-0.3 (0.12)</td>
<td>1-3 (2.2)</td>
<td>6.8-14.5 (8.2)</td>
</tr>
</tbody>
</table>

4 hrs. | Spike discharge disappeared.

Before occlusion

5 minutes after occlusion

20 minutes

1 hour

2 hours

Fig. 2. Changes in EMG following occlusion of the mesenteric vessels.

occlusion, the spike discharges became quite weak and irregular, showing abortive spikes and slow waves, and at four hours electric discharges of any pattern were no longer recognized.

In the case of an incomplete vascular occlusion that did not cause a prompt change in color of the involved intestine, the action potential was perceptible even after two hours or later. Also on this occasion, however, the
intestine fell into electrical standstill in about six hours.

3. EMG during strangulating obstruction

Strangulating obstruction of the intestine was induced in eight dogs of this series. On gross observation, a complete strangulation was followed almost immediately by an increase in the peristaltic activity of the involved intestine. The tint of the strangulated loop changed from darkish red to reddish purple in approximately 20 minutes. At the same time, petechiae appeared on the serosal surface of the involved intestine and mesentery, and hemorrhagic exudate began to accumulate in the peritoneal cavity. Two to three hours later, the movement gradually became less active but the resting interval was never noted in this instance. The intestinal motility in the case of complete strangulation thus resembled that of vascular occlusion. On the other hand, when strangulation was incomplete and the intestine became only dark red in color, the peristaltic movement was hyperactive for more than two hours and the resting interval appeared at about four hours, thus resembling the finding in the case of mechanical obstruction of the intestine.

In this group, the electromyographic findings obtained at the intestinal segment oral to the strangulated loop were omitted because of their resemblance to those in the case of simple mechanical obstruction. Table III summarizes the findings at the strangulated loop in the case of complete strangulation and Fig. 3 demonstrates EMG of a case of this group. In 20 to 30 minutes after onset of complete strangulation, the spike number and the duration of the spike burst increased much and the interval between bursts was shortened considerably, although there was no significant change in the amplitude of the burst. At 30 to 120 minutes, the increases in the spike number and in the duration of bursts became more remarkable and the amplitude of bursts became apparently longer than before strangula-

<table>
<thead>
<tr>
<th>Time since onset of strangulation</th>
<th>Maximum amplitude (mV)</th>
<th>Mean amplitude (mV)</th>
<th>Duration (sec.)</th>
<th>Spike number</th>
<th>Interval between bursts (sec.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before strangulation</td>
<td>0.8-1.7 (1.2)</td>
<td>0.6-1.4 (1.0)</td>
<td>0.1-0.4 (0.21)</td>
<td>1-4 (1.7)</td>
<td>2.2-3.4 (2.5)</td>
</tr>
<tr>
<td>20-30 min.</td>
<td>0.8-1.4 (1.25)</td>
<td>0.6-1.2 (1.0)</td>
<td>0.5-0.8 (0.57)</td>
<td>5-10 (7.7)</td>
<td>1.6-2.2 (1.83)</td>
</tr>
<tr>
<td>30-120 min.</td>
<td>1.3-2.8 (2.5)</td>
<td>1.2-2.3 (2.1)</td>
<td>0.4-0.8 (0.61)</td>
<td>7-12 (8.7)</td>
<td>1.8-5.8 (2.8)</td>
</tr>
<tr>
<td>2-4 hrs.</td>
<td>0.3-0.7 (0.46)</td>
<td>0.2-0.5 (0.37)</td>
<td>0.2-0.4 (0.3)</td>
<td>4-8 (5.9)</td>
<td>7.8-10.8 (8.8)</td>
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<tr>
<td>More than 6 hrs.</td>
<td></td>
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<td>Spike discharge disappeared.</td>
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</table>
Fig. 3. Changes in EMG during complete strangulation.

At about two hours after onset of strangulation, the spike bursts began to decrease in the amplitude, spike number and duration. At the same time, the interval between bursts, which had been quite uniform at 30 to 120 minutes, became more and more irregular and prolonged, and the intestine became electrophysiologically inert in four to six hours. However, the resting interval was never observed in EMG of this group, and in this sense complete strangulation resembled vascular occlusion.

The comparable results in the case of incomplete or mild strangulation are shown in Table IV and Fig. 4. In 60 to 90 minutes, the amplitude, the duration and the spike number of the burst increased much and the interval between bursts was significantly shortened, indicating intensification of the peristaltic movement. Such an increase in the electrophysiologic activity of the intestine was still...
remarkable in four to six hours after onset of strangulation, but a tendency toward grouping of spike bursts, and an appearance of the resting interval was observed in this period; a group of spike bursts consisting of 13 to 18 individual bursts and a resting interval lasting 60 to 84 sec. began to appear alternately. Accordingly, EMG in this stadium had some resemblance with those in the case of simple mechanical obstruction. The action potential gradually diminished thereafter and disappeared in about 18 hours after onset of strangulation.

4. EMG during recovery from intestinal obstruction

1) Recovery from mechanical obstruction

When the intestine was released from mechanical obstruction after three hours’ duration, i.e., in the early phase where the obstructed intestine was slightly distended showing hyperactive peristalsis and the EMG exhibited intensified spike discharges, the abnormal hyperactivity of the gross movement as well as that of electric discharges soon began to subside and the EMG resumed to show an approximately normal pattern in about 30 minutes, as indicated in Fig. 5, A and Table V, A.

When the removal of obstruction was done after eight hours, i.e., in the intermediate phase where the gross and electromyographic evidence of alternation of a strengthened peristaltic movement and a resting interval was remarkable, the condition began to normalize gradually and the EMG became approximately normal in an hour, as shown in Fig. 5, B and Table V, B.

When obstruction was removed after 24 hours or later, i.e., in the late phase
A) Removal of the cause in the early phase

Before removal

30 minutes after removal

B) Removal of the cause in the intermediate phase

Before removal

1 hour after removal

Fig. 5. Changes in EMG in the course of recovery from mechanical obstruction.

characterized by a marked distention of the intestine, disappearance of grossly recognizable peristaltic movement and absence of electric discharges, the peristalsis and the spike discharges did not appear for a while and it seemed as if obstruction had still continued. With a decrease in the content of the intestinal loop, a feeble peristaltic movement became recognizable and EMG showed a trace of spike discharges. However, the intestinal movement and the electrophysiologic activity were not restored completely and the animals expired within the next 24 hours.

2) Recovery from vascular occlusion

When the clamp occluding the mesenteric vessels was removed after 30
minutes, i.e., while the peristaltic movement was hyperactive, the involved intestine recovered to normal in appearance in about five minutes. In accordance with this, the hyperactivity in spike discharges was corrected and the EMG resumed a normal pattern in about five minutes, as shown in Fig. 6, A and Table VI, A.

When the circulation was re-established after two hours' duration of vascular occlusion, i.e., in the period in which EMG was characterized by sporadic appearance of weak spike bursts, the intestine changed from reddish purple to fresh pink, the tonic contraction subsided and the peristaltic movement appeared in several minutes. The EMG showed evidence of hyperperistalsis after five

<table>
<thead>
<tr>
<th>Table V. Electromyographic Findings after Removal of Mechanical Obstruction</th>
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<tr>
<td>------------------------</td>
</tr>
<tr>
<td>A. Removal in the early phase</td>
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<td>B. Removal in the intermediate phase</td>
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<table>
<thead>
<tr>
<th>Table VI. Electromyographic Findings after Removal of Vascular Occlusion</th>
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<tr>
<td>------------------------</td>
</tr>
<tr>
<td>A. Removal 30 minutes after occlusion</td>
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<tr>
<td>B. Removal 2 hours after occlusion</td>
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minutes and became approximately normal in about 30 minutes, as shown in Fig. 6, B and Table VI, B. However, an irregularity in the interval between bursts was

A) Removal of the cause 30 minutes after occlusion

Before removal

5 minutes after removal

B) Removal of the cause two hours after occlusion

Before removal

5 minutes after removal

30 minutes after removal

Fig. 6. Changes in EMG in the course of recovery from vascular occlusion.

still observed 30 minutes after removal of the clamp.

At more than three hours after onset of vascular occlusion, the involved intestine became edematous and dark red in color, and showed petechiae and hemorrhagic exudate, as mentioned before. When the circulation was re-established at that time, the tint of the intestine did not show the sign of recovery, and peristalsis and spike discharges were not observed even after several hours.
DISCUSSION

In 1922, Alvarez and Mahoney\textsuperscript{3}) succeeded for the first time in recording the action potential of the rabbit intestine. Subsequently, Bozler\textsuperscript{4}) noticed that the peristaltic movement of the intestine was accompanied by spike discharges. It was then elucidated by Takita\textsuperscript{5}) and by many other investigators that action currents led from the intestine consisted of spike bursts which appeared at certain intervals and that the number of spikes, duration and amplitude of the burst increased with an increase in intensity of the peristaltic movement. Electromyography has thus become quite useful in the study of motor function of the intestinal tract.

There are many reports on the intestinal motility in intestinal obstruction but those dealing with the problem by means of electromyography are few. In this connection, the present study was undertaken to analyze intestinal electromyograms (EMG) during intestinal obstruction in dog. Simple mechanical obstruction and obstruction due to vascular occlusion were studied separately and strangulating obstruction was also subjected to the study.

\textit{Mechanical obstruction.} In the early phase (first eight hours) of mechanical obstruction, EMG revealed an increased activity of peristaltic discharges, i.e., increases in the spike number, duration and amplitude of spike bursts. Such hyperperistalsis became more remarkable in the intermediate phase (8 to 12 hours) and, at the same time, a resting interval showing no spike discharges began to appear, so that EMG in this period was characterized by an alternate appearance of hyperactive and resting intervals. In the late phase (after 12 hours or later), the EMG revealed decreases in the spike number, duration and amplitude of spike bursts and the resting interval was prolonged much, eventually leading to disappearance of peristaltic discharges.

When the reports on the intestinal movement during simple mechanical obstruction are reviewed, it seems to be generally accepted that a kind of hyperperistalsis, so-called "stenotic movement", occurs in an early phase at the portion oral to the site of obstruction but the involved intestine eventually undergoes paralysis for excessive stretch of its blood vessels due to intestinal distention. Shiokawa\textsuperscript{6}) investigated the frequency of peristalsis during intestinal obstruction in rabbits and dogs, and found that the frequency became maximum approximately four hours after onset of obstruction but apparently it began to decrease after 10 hours. Nishijima\textsuperscript{2}) simultaneously recorded EMG and mechanogram of the rabbit intestine in the case of mechanical obstruction and observed that hyperactive peristaltic waves associated with increases in the amplitude, spike number and duration of spike bursts appeared in the first 15 hours. The electromyographic findings in the early phase of the present experiment thus quite resemble those obtained by Nishijima in rabbits.
On the other hand, Takita et al.\textsuperscript{1) observed in a stadium of mechanical obstruction an alternate incidence of hyperactive peristalsis and an interval in which the intestine appeared to be inactive. Such a phenomenon became prevalent about 24 hours after onset of obstruction and they named this phenomenon as “vicissitude type movement”. In his simultaneous observation of EMG and mechanogram, Nishijima\textsuperscript{2) also noted a similar phenomenon that became remarkable at 12 to 15 hours after onset of mechanical obstruction. In the present experiment, an alternation of a group of spike bursts and a resting interval was observed at 8 to 12 hours after induction of mechanical obstruction, which may be identical with what was described by Takita et al. According to Asao\textsuperscript{7) who recorded by Magnus’ method the motility of a strip of muscle tissue isolated from the intestinal wall affected by mechanical obstruction, such a muscle strip exhibited an alternate repetition of contraction and relaxation. Thus it is likely that the vicissitude type movement is due to a change in the motility of muscle fibers, probably representing a physiological adaptation of the fibers to an increased requirement of peristalsis.

In the late phase of mechanical obstruction, the author observed a curious phenomenon that the distended intestine showed a long-durating tonic contraction without exhibiting any electric discharges. Such a phenomenon was formerly pointed out by Bozler\textsuperscript{8) who reported the instances in which the action potential could not be recorded despite the presence of so strong a contraction ring as to make the intestine pale in appearance. Takita\textsuperscript{5) also observed a periodic appearance of long-lasting intensive contractions in the intestine at a late stage of mechanical obstruction and found such periodic contractions to be spasms or pathological contractions unrelated to spike discharges. Besides such a phenomenon, the present experiment also revealed an occasional appearance of feeble electric discharges in the extremely distended intestine that did not show grossly appreciable movements.

\textit{Vascular occlusion.} In the present experiment, occlusion of the mesenteric vessels was shortly followed by increases in the spike number, duration and interval between bursts of EMG of the involved intestine. Such intensification of spike bursts showed an acme approximately 20 to 30 minutes later and became less remarkable thereafter, the discharges disappearing in about two hours. Concerning the intestinal movement during circulatory disturbances, it seems to be a general idea that the peristaltic movement is rather inhibited on such occasion. However, a number of experimental facts indicate an acceleration of peristalsis in circulatory disturbances, or anoxic conditions of the intestine. In 1944, Van Lier\textsuperscript{9,10) withdrew blood from the femoral artery of dog and observed hyperperistalsis of the intestine six hours later. Since hyperperistalsis of the intestine at that time was inhibited by intravenous administration of cocain, an activator of epinephrin, he concluded that anoxia due to exsanguination might have stimulated
the intestinal movement through the parasympathetic system rather than through the sympathetic system. Shiratori\textsuperscript{11}) was also able to induce hyperperistalsis of the gastrointestinal tract by exsanguination. According to Fukuda\textsuperscript{12}), the peristaltic movement of the small intestine was also accelerated in the case of anoxic anoxia caused by inhalation of oxygen and nitrous oxide mixture. As regards stagnant anoxia, Shiratori\textsuperscript{13}) reported that the blood vessels distributed to the lesser curvature of the stomach were intimately related to the motor function of the stomach and their occlusion resulted in hyperactive movements of that organ. The result of the present study that an intensive peristalsis occurred in the intestine shortly after occlusion of the mesenteric vessels agrees with these previous observations.

Thus, comparing the EMG in the case of vascular occlusion with that of mechanical obstruction, the author could point out both resemblance and difference. The point of resemblance is that the spike discharges are first intensified and then weakened. The difference, on the other hand, may be reduced to the following two points: Firstly, the mentioned change in EMG progresses more rapidly in the case of vascular occlusion than in mechanical obstruction. Secondly, the EMG in mechanical obstruction is characterized, especially in later stages, by an alternate appearance of intensified spike bursts and a resting interval, while the resting interval is never observed in EMG in vascular obstruction.

\textit{Strangulating obstruction}. Strangulating obstruction includes vascular occlusion besides mechanical obstruction of the intestinal lumen. The vascular occlusion in this case is usually classified into the arterial and the venous types according to the principally involved vessel and into the complete and the incomplete types according to its severity. When the mesenteric vessels are occluded, a change in the capillary permeability is induced and results in stagnant edema and hemorrhagic exudation. In this mechanism, the venous type is believed to be severer than the arterial one.

Only a few reports have been presented concerning the electromyographic aspect of the intestinal motility in the case of strangulating obstruction. Nishijima\textsuperscript{2}) observed changes in the intestinal EMG during the course of strangulation of the rabbit intestine and reported that the amplitude, spike number and duration of spike bursts increased much in the first two hours but then they tended to decrease, showing only a trace of discharges after six hours. On gross observation of the intestinal movement in the case of experimental strangulation of the rabbit intestine, Ooshima\textsuperscript{14}) described a repetition of strong peristalsis at an interval of five to six seconds, an occurrence of grouped peristaltic rings followed by a resting interval, and a sporadic appearance of a continuous contraction occurring every five to ten seconds. The author’s observation is summarized as follows: In the case of complete strangulation, the EMG resembled that in the case of vascular occlusion, revealing increases in the amplitude,
spike number and duration of spike bursts for the first two hours and
debilitation of spike bursts thereafter. In the case of mild strangulation, on the
other hand, manifestations of mechanical obstruction were predominant in EMG,
a group of intensified spike bursts and a resting interval appearing alternately.
These results have some resemblance with the observations by Nishijima\textsuperscript{2}) and
Ooshima\textsuperscript{14}) in that a strangulation of the intestine is temporarily followed by
hyperperistalsis. However, the present study has also clarified an interesting
aspect of strangulating obstruction that the EMG on strangulation of the intes-
tine includes manifestations of both mechanical obstruction and vascular occlu-
sion and which one is predominant is dependent upon the severity of strangula-
tion.

\textbf{Recovery from intestinal obstruction.} When the cause of intestinal obstruc-
tion is removed within certain duration, the affected intestine is restored from
obstructive conditions. However, few reports have dealt with the electromyo-
graphic aspect of the recovery process. Saito\textsuperscript{15}) experimentally produced mechanical
obstruction of the dog or rabbit intestine by ligation with silk and removed
the ligation after the proximal intestine had shown marked distention. The
recovery on this occasion was very slow and the intestine appeared for a consider-
ably long period as if obstruction had been still remaining. Complete recovery
was attained first after a week. Ooshima\textsuperscript{14}) observed that two hours after removal
of the cause of strangulating obstruction, the tint of the intestine became reddish
and vivid, but peristaltic movements were interrupted or weakened at the portion
where strangulation had occurred. These two reports more or less emphasize a
fairly long duration of obstructive conditions after removal of the cause of
obstruction. According to Inose\textsuperscript{14}), degeneration of the nervous plexus in the
intestinal wall occurs in a comparatively early phase of intestinal obstruction and
it is estimated that a disturbance of the autonomic nervous system is a cause of
irreversible dysfunction of the intestine in the case of intestinal obstruction.
Fukuhara\textsuperscript{17}) observed that when the intestinal segment was perfused with Tyrode's
solution to make it completely ischemic, the nervous ganglia in the wall of the
involved intestine were destroyed within four hours. These two reports suggest
that the chance for recovery of the motor function of the intestine from intestinal
obstruction is largely dependent on the grade of damage of the autonomic nervous
system or the smooth muscle of the affected intestine.

The present results indicated that the intestine involved by intestinal obstruc-
tion could be restored to an electromyographically normal condition if the cause of
obstruction was removed within a limited time. In the case of mechanical
obstruction, the recovery of the intestinal movement from the early phase of
obstruction was quite easy; that from the intermediate phase required a longer
time; and that from the late phase was never attained. In the case of vascular
occlusion, the intestinal movement returned to normal in 10 to 30 minutes when
circulation was re-established within two hours after onset of the occlusion, but the movement never recovered to normal when circulatory disturbances lasted for more than two hours. The maximum duration of intestinal obstruction permitting recovery of the normal motor function of the intestine was thus estimated in this series to be approximately 24 hours in the case of mechanical obstruction and about two hours in the case of vascular occlusion. This is quite interesting in relation to the clinical fact that patients with intestinal obstruction occasionally die within two or three days after surgical removal of the cause of obstruction.

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