Electrophysiological Studies of the Antrum Muscle Fibers of the Guinea Pig Stomach

H. KURIYAMA, T. OSA, and H. TASAKI

From the Department of Physiology, Faculty of Medicine and Dentistry, Kyushu University, Fukuoka, Japan

ABSTRACT The membrane potentials of single smooth muscle fibers of various regions of the stomach were measured, and do not differ from those measured in intestinal muscle. Spontaneous slow waves with superimposed spikes could be recorded from the longitudinal and circular muscle of the antrum. The development of tension was preceded by spikes but often tension appeared only when the slow waves were generated. Contracture in high K solution developed at a critical membrane potential of −42 mv. $MnCl_2$ blocked the spike generation, then lowered the amplitude of the slow wave. On the other hand, withdrawal of Na^+, or addition of atropine and tetrodotoxin inhibited the generation of most of the slow waves but a spike could still be elicited by electrical stimulation. Prostigmine enhanced and prolonged the slow wave; acetylcholine depolarized the membrane without change in the frequency of the slow waves. Chronaxie for the spike generation in the longitudinal muscle of the antrum was 30 msec and conduction velocity was 1.2 cm/sec. The time constant of the foot of the propagated spike was 28 msec. The space constants measured from the longitudinal and circular muscles of the antrum were 1.1 mm and 1.4 mm, respectively.

INTRODUCTION

The early investigations of mammalian stomach muscle suggested that membrane activity consisted of spike and slow wave components (Alvarez and Mahoney, 1922; Richter, 1923; Bozler, 1938, 1942; and Ichikawa and Bozler, 1955; Daniel, 1965). In 1945, Bozler studied the stomach of the dog, cat, and guinea pig, using nonpolarizable differential electrodes consisting of two capillary calomel electrodes and recorded the differential potential associated with each peristaltic contraction. Three main waves, designated R, S, and T, were described in the dog's stomach. The shape of the action potential was identical with that of some other visceral muscles and of cardiac muscle but the complex lasted for 5–8 sec. In the guinea pig stomach, Bozler observed

that a slow potential change could be observed only in the pyloric region, whereas in the middle portion of the stomach the discharge consisted of brief spikes only. Ichikawa and Bozler (1955) confirmed the above results for the dog and came to the conclusion that the smooth muscle of the stomach was a single muscular unit resembling that of cardiac muscle. Kolodny and Van der Kloot (1961) reported that the membrane activity of the frog stomach muscle could be recorded as repetitive spike discharges, even in sodium-free sucrose solution. Berger (1963) observed the membrane activity of frog stomach using the double sucrose gap method, and reported that the circular muscle showed only slow potential changes.

Recently, Papasova, Nagai, and Prosser (1968) studied the cat stomach muscle using a pressure electrode and reported that cat stomach in vitro showed spontaneous slow waves and spikes and that the slow waves consisted of sodium-dependent and calcium-dependent components. Both components of the slow waves were thought to originate in longitudinal muscle.

Electrophysiological studies on the guinea pig stomach using microelectrode techniques have not yet been done systematically. The present investigation was carried out to observe the electrical specificity of the muscle fibers of the guinea pig stomach mainly in the region of the antrum, and to compare the results with those obtained in the guinea pig intestinal smooth muscle.

METHODS

Guinea pigs, weighing 250–300 g, were stunned and bled. The stomach was excised and dissected, starting along the greater curvature. Connective tissue was carefully removed under Krebs solution at room temperature. The muscle layers were separated from the mucous membrane, and the longitudinal muscle layer was fixed on a rubber plate. The microelectrode was inserted from the serosal side. When the circular muscle was studied, the microelectrode was inserted from the mucosal side. The tissue was mounted in an organ bath through which solution flowed continuously at a temperature of 35–36°C. A modified Krebs solution of the following composition was used (mM): Na^+ 137.4; K^+ 5.9; Mg^{++} 1.2; Ca^{++} 2.5; Cl^- 134.0; HCO_3^- 15.5; $H_2PO_4^-$ 1.2; and glucose 11.5; equilibrated with 97% O_2–3% CO_2.

Unipolar stimulation was used to elicit spikes. The electrodes consisted of an Ag-AgCl needle electrode (diameter 0.5 mm) placed on the tissue with another electrode placed at a distance of 3 cm from the tissue. In order to measure the passive electrical constants of the smooth muscle, the same microelectrode was used for intracellular recording as well as for stimulating by means of the Wheatstone bridge method described by Kuriyama and Tomita (1965). In order to insure a constant current into the cell, a much higher resistance (1000 M) than that of the microelectrode was used as one bridge arm in series with the microelectrode. The range of applied current was between 10^{-10} and 5×10^{-9} amp. The error introduced by a possible resistance change of the electrode during the experiment was probably less than 10%. To measure the time constant of the membrane by extracellularly applied

electrical current, Ag-AgCl plate electrodes were used as described by Tomita (1966 *a*, *b*).

Isometric tension was recorded simultaneously with membrane activity using a strain gauge (Nihon Kohden Ltd.). One side of the preparation was fixed on the rubber plate, and the other side was tied by silk thread and connected to the hook of the tension recorder.

To measure conduction velocity and to observe the synchronization between membrane activity at different sites, the recording electrodes were inserted into two different cells and the distance between the two electrodes was measured under a binocular microscope. The concentration of drugs used is described in the results.

TABLE I

MEMBRANE POTENTIAL MEASURED FROM VARIOUS PARTS OF THE GUINEA PIG STOMACH

Tissue	Membrane potential	SE	Nos. (*n*)	Overshoot potential
	−*mv*	±*mv*		
Longitudinal muscle				
Fundus	58.1	1.2	55	+
Corpus				
Greater curvature	60.1	0.3	130	+
Lesser curvature	59.3	1.0	50	+
Antrum	60.4	0.8	50	+
Pylorus	61.2	1.1	35	+
Circular muscle				
Fundus	57.4	0.7	42	+
Antrum	58.2	0.8	50	+
Pylorus	54.1	0.9	50	+

The principal drugs used were tetrodotoxin (Sankyo Pharmacological Co., Ltd., Tokyo, Japan), atropine sulfate, acetylcholine chloride, prostigmine methylsulfate, epinephrine hydrochloride, and 5-hydroxytryptamine.

RESULTS

General Properties of the Membrane

Table I shows the membrane potentials measured from the various parts of the stomach. The resting membrane potentials of the stomach varied from −54.1 mv to −61.2 mv. These values were slightly higher than those observed in the guinea pig intestine (Holman, 1958; Bülbring, 1962; Bülbring and Kuriyama, 1963; Kuriyama, Osa, and Toida, 1967 *b*, *c*).

Spontaneous discharges could be recorded from all regions of the longitudinal and circular smooth muscle layers of the stomach. Usually, the spontaneous discharges appeared as repetitive spike discharges superimposed on a

slow wave of depolarization. The amplitudes of the slow wave varied for individual fibers from a few millivolts to 40 mv, and the duration of the slow wave ranged from 5 to 12 sec. The slow depolarization could elicit a spike as a generator potential, although the critical firing level or threshold membrane potential, for spike generation was not constant. The amplitude of successive spikes in a train gradually fell in proportion to an increasing depolarization of the slow wave. When the slow wave showed a sustained depolarization, spikes became abortive and often developed into an oscillation. During the repolarization phase of the slow wave, the spike amplitudes were again gradually enhanced. Sometimes, spontaneous discharges of spikes appeared without a slow potential change, when the membrane potential was low (−40 to −45 mv), e.g. after perfusion in the Krebs solution for several hours.

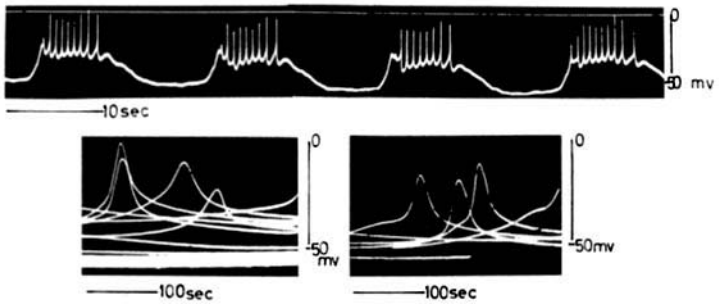

FIGURE 1. Typical pattern of the spontaneous membrane activity recorded from the longitudinal muscle of the antrum. Upper trace, continuous recording; lower trace, superimposed photograph with rapid sweep.

Fig. 1 shows a typical pattern for spontaneous membrane activity recorded from the longitudinal muscle of the antrum. In this cell, the maximum resting membrane potential during the quiescent period was −62 mv and the slow potential changes appeared at regular intervals of about 8 sec. The first spike in a discharge appeared when the membrane was depolarized to −48 mv. The maximum rates of rise and fall of the spike recorded from the longitudinal muscle of the antrum were 5.5 v/sec (SE = ±0.03, n = 25) and −7.6 v/sec (SE = ±0.7, n = 25), respectively. These values were much lower than those observed in the guinea pig intestine (15 v/sec, Holman, 1958; Bülbring and Kuriyama, 1963).

Relation between Membrane Activity and Tension Development

Fig. 2 shows the relation between spikes and tension development recorded from the longitudinal muscle of the antrum. In Fig. 2 a single spike triggered phasic contraction, and repeated spikes elicited summated contraction (upper trace). In some cells, the slow wave preceded phasic tension develop-

ment (lower trace). However, this phenomenon does not indicate that the tension development was triggered by the slow wave, as the membrane activity was recorded from a single cell, while the tension was recorded from the whole tissue.

The membrane potential and tension development were measured from

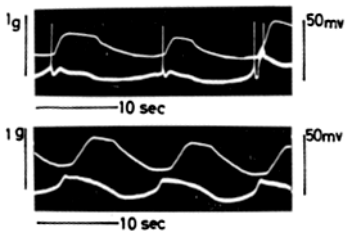

FIGURE 2. Simultaneous recordings of the membrane activity and tension from the longitudinal muscle of the antrum. Upper trace, spike and tension development; lower trace, slow potential change and tension development.

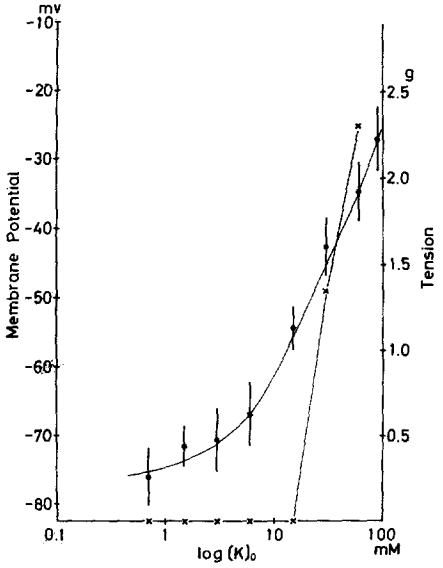

FIGURE 3. Effects of various external potassium concentrations on the membrane potential recorded from the longitudinal muscle of the antrum and tension development. Vertical lines indicate twice the standard deviation. Solid circles indicate membrane potential and x indicates tension development.

the longitudinal muscle of the antrum in the presence of different external potassium concentrations. The tissue was pretreated with 10^{-6} g/ml atropine and 10^{-6} g/ml tetrodotoxin to exclude nervous activity. Furthermore, the perfusing solution was cooled to 28°C in order to reduce the membrane activity in high $[K]_o$. Fig. 3 shows the relationship between the membrane potential and tension development of the greater curvature of the stomach at various external potassium concentrations. 2.5 times the normal potassium concentration (14.5 mM) depolarized the membrane from −66.9 mv (SE = ±0.8, n = 30) to −54.4 mv (SE = ±0.9, n = 35). However, no tension development was observed. When the potassium concentration was increased

to 29.4 mM, tension development (contracture) could be observed. The critical membrane potential to evoke contracture was about −42.4 mv (SE = ±0.7, n = 32). As described above, the slow wave developed from a few millivolts to 40 mv at the membrane potential of about −60 mv. Therefore, the maximum amplitude of the slow wave exceeded the critical membrane potential needed to evoke the contracture.

When a small piece of stomach muscle was excised from the antrum near the greater curvature, slow waves of large amplitude often appeared without spikes, or with only a few spikes. In contrast, specimens excised from the pylorus generated slow waves of lower amplitude than those seen in cells of the greater curvature, but with a higher frequency of spike discharge. The

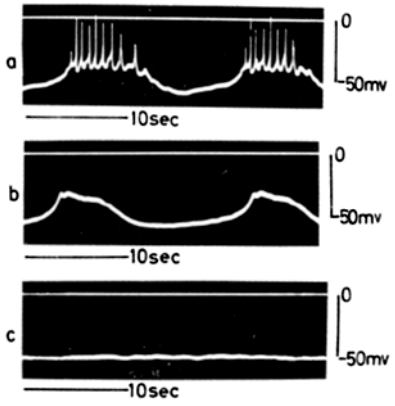

FIGURE 4. Effects of MnCl (1 mM) on the longitudina$_2$ smooth muscle cell of the stomach (antrum). a, control; b, treatment with $MnCl_2$ after 5 min; c, after 15 min.

phasic changes in tension appeared more consistently in the pyloric region than in the other parts of the stomach.

When a microelectrode was inserted in the circular muscle of the antrum, the slow wave and superimposed spikes appeared more consistently than when recording from the longitudinal muscle. The slow wave and spikes could also be recorded from the dissected pyloric circular muscle layer (Kuriyama, Sakamoto, and Tomita, data to be published).

Drug Action on the Spike and Slow Wave

When membrane slow potential changes were recorded from the longitudinal muscle of the antrum, $MnCl_2$ (1 mM) in the Krebs solution first blocked the spike generation but the amplitude of the slow wave was only partially reduced as shown in Fig. 4. After more than 15 min of exposure, both the spike and the slow wave were completely blocked. More than 2 mM of $MnCl_2$ rapidly blocked both the spike and the slow wave.

Actions on the spike and slow wave opposite to those observed with $MnCl_2$ have been observed after treatment either with tetrodotoxin or Na-free (substituted for by Tris) solution.

In five out of seven specimens, tetrodotoxin (10^{-7} g/ml) did not block spontaneous spike generation but gradually lowered the amplitude of the slow wave. The number of spikes in a train discharge was reduced in accordance with a decrease in the amplitude of the slow wave. Tetrodotoxin (10^{-6} g/ml) blocked the generation of the slow waves and also the spontaneously generated spikes.

Electrical current extracellularly applied, however, elicited a spike from the muscle after treatment with tetrodotoxin. The amplitude of the spike remained the same as that before the treatment. More detailed studies of the spike generation mechanism under various ionic conditions will be described in another paper (Kuriyama, Sakamoto, and Tomita, data to be published).

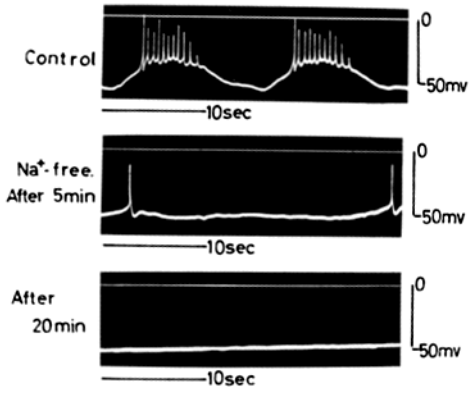

FIGURE 5. Effect of Na-free solution on the membrane activity of the longitudinal muscle of the antrum. The Na-free solution was prepared by substitution of Tris-Cl for NaCl and $NaHCO_3$. pH was adjusted to 7.3.

Fig. 5 shows the effect of Na-free (Tris) solution on the membrane activity. Na-free solution first reduced the amplitude of the slow wave, then blocked spike generation. However, even without generation of the slow wave, the pacemaker type of spike which was preceded by a prepotential could be recorded although the spontaneously generated spike itself often ceased after treatment with either tetrodotoxin (10^{-6} g/ml) or Na-free solution. Electrical stimulation of the tissue, however, elicited the spike in the above solutions, but never in the presence of $MnCl_2$ (1 mM).

In order to investigate whether the slow potential change is related to cholinergic transmission, the effects of acetylcholine, prostigmine, and atropine on the slow wave were examined. Acetylcholine (10^{-6}–10^{-5} g/ml) depolarized the membrane but did not reduce the duration of the silent period between bursts of spontaneous activity. The spike frequency on the slow wave was not influenced even though the membrane was depolarized more than 20 mv (10^{-5} g/ml of acetylcholine).

Fig. 6 shows the effects of acetylcholine (10^{-6} g/ml and 10^{-5} g/ml) on the membrane activity of the longitudinal muscle of the antrum. Compared with the intestine this tissue showed very low sensitivity to acetylcholine. For ex-

ample, the longitudinal muscles of the jejunum and cecum respond to 10^{-8}–5×10^{-8} g/ml of acetylcholine which depolarize the membrane thus increasing the spike frequency.

Prostigmine (10^{-7}–10^{-5} g/ml) was more effective than acetylcholine. Fig. 7 shows that prostigmine (10^{-6} g/ml) depolarized the membrane of the longi-

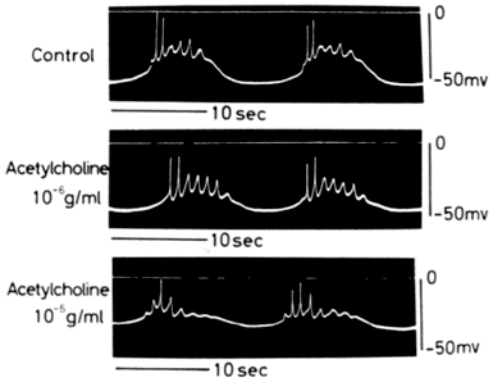

FIGURE 6. Effects of acetylcholine (10^{-6} and 10^{-5} g/ml) on the membrane activity of the longitudinal muscle of the antrum.

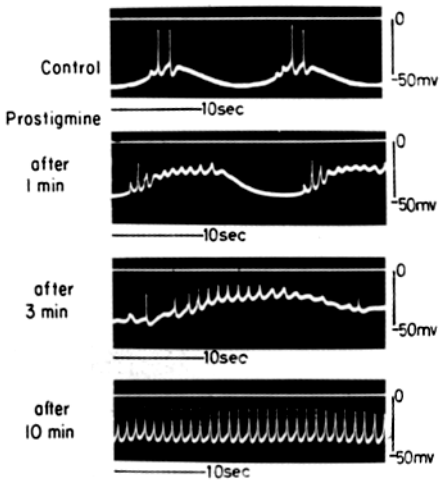

FIGURE 7. Effects of prostigmine (10^{-6} g/ml) on the spontaneous membrane activity of the longitudinal muscle of the antrum. Control, after 1 min, 3 min, and 10 min. Horizontal bars indicate the zero potential level.

tudinal muscle of the antrum, enhanced the amplitude of the slow wave, reduced the duration of the silent period, and increased the number of spikes in a train. Furthermore, after long exposure to prostigmine (10^{-8}–10^{-6} g/ml) the membrane became depolarized from -50 mv to -35 mv which caused continuous generation of spikes with an abortive shape.

Atropine (10^{-7} g/ml) hyperpolarized the membrane by 5–10 mv. When the concentration of atropine was increased to 10^{-6} g/ml, slow waves ceased completely in three out of five specimens. However, in one specimen, atropine (10^{-6} g/ml) had no effect at all on the slow potential.

Response of the Membrane to the Electrical Stimulation

Extracellular field stimulation (0.5 mm diameter Ag-AgCl electrode, 0.2–0.5 msec pulse duration) to the longitudinal muscle of the stomach caused spikes and slow waves. Comparison of the slow wave elicited by the field stimulation and the spontaneously generated one showed that the duration of the elicited response was only 200 msec and repetitive spikes never appeared on it. Fig. 8 shows responses of the longitudinal muscle of the greater curvature (antrum) to field stimulation (0.5 msec pulse duration). The recording electrode was placed 1 mm away from the stimulating electrode. In this particular cell (a), the spike was not elicited as a response to the electrical stimulation but was

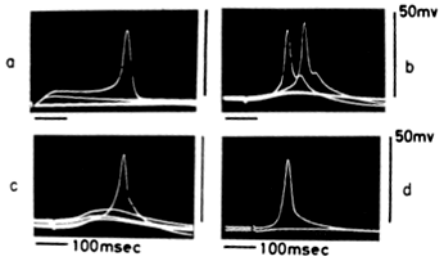

FIGURE 8. Various types of response of the longitudinal muscle of the stomach (antrum) to field stimulation (5–10 msec of pulse duration). a, b, c, and d were recorded from the different cells. d was recorded after treatment with atropine (10^{-5} g/ml).

produced by the depolarization of the membrane with a long latency. When weaker stimulation was applied to the tissue, only the slow wave was recorded (a, b, and c). A spike could also be elicited without generation of the slow wave (d), i.e. after treatment with atropine (10^{-5} g/ml), the membrane could generate a spike in response to strong field stimulation.

The above results, namely, the effect of tetrodotoxin, responses to cholinergic drugs (atropine, acetylcholine, and prostigmine), and the response of the membrane to field stimulation indicate that the slow waves generated in the stomach may be due to nerve stimulation, and some of the components were presumably due to a release of acetylcholine from the cholinergic nerves distributed in the muscle layers. However, the slow potentials of some specimens had not been influenced by treatment with tetrodotoxin or atropine. Therefore, we conclude that generation of the slow potential was not due only to nervous factors.

Extracellular field stimulation (1–5 msec pulse duration) sometimes elicited inhibitory potentials with a latency of 50–120 msec. The inhibitory potential stopped the spontaneous discharges, as observed in the intestine. However, generation of the inhibitory potential was rare compared with that of the excitatory potential.

The chronaxie for the spike of the longitudinal muscle of the antrum elicited by extracellular stimulation varied from 20 msec to 38 msec (the mean value was 30 msec, $n = 5$).

The chronaxie for the spike generation of the smooth muscle might give the time constant of the membrane if the cable theory is applicable to the stomach muscle (chronaxie = time constant $\tau \times 0.22$; cf. Tomita, 1966 *a*, *b*). The chronaxie was 30 msec, which would correspond to a time constant of the membrane of 140 msec. This value is in rough agreement with that measured by the extracellular polarizing method.

The conduction velocity varied from 0.6 cm/sec to 1.9 cm/sec (the mean value was 1.2 cm/sec, $n = 6$). These values were measured by the insertion of two microelectrodes, 1 mm apart, and the stimulating electrode was placed 5 mm away from the first recording electrode.

The time course of the foot of a propagated action potential of the longitudinal smooth muscle fibers of the antrum was measured. The distance between the extracellular stimulating electrode and the recording microelectrode was about 5 mm along the greater curvature. The foot of the spike rose exponentially and its mean time constant was 28 msec in eight experiments (SE = ± 1.4). Spikes were chosen from the records which were not triggered by slow changes in membrane potential (one measurement was done from Fig. 8 *d*).

A large membrane capacitance could be expected from the large value of the time constant of the foot of the propagated spike. The time constant of the foot of the spike is much less (7 msec) in the guinea pig taenia coli (Tomita, 1966).

Passive Membrane Properties of the Antrum

The input resistance (R_{eff}) and the time constant (τ) were measured for the longitudinal muscle fiber of the greater curvature of the antrum by the application of weak hyperpolarizing current through a microelectrode. The input resistance varied from 10 MΩ to 43 MΩ (mean input resistance was 24 mΩ, SE = ± 2.0, $n = 21$). The time for 85% decay of the electrotonic potential was 2.3 msec (SE = ± 0.07, $n = 20$). The time courses of these electrotonic potentials were much faster than those of the electrotonic potentials set up by extracellular current-passing electrodes (see below).

When a current-voltage relation was observed by applications of intracellular polarizing currents, depolarizing currents evoked a slightly lower amplitude of electrotonic potential than those observed with hyperpolarizing current at any given intensity; i.e., it was possible to recognize weak rectification of the membrane.

On the other hand, the time constant for the longitudinal muscle membrane of the antrum (greater curvature side) measured by the extracellular

polarizing method described by Tomita (1966 *b*) was 160 msec (n = 5) at the time of 85% decay of the electrotonic potential (i.e. decay to 15% of steady potential state). The circular muscle also showed nearly the same value for the time constant of the membrane (180 msec, n = 6) under the same conditions as those measured for the longitudinal muscle. These potential changes were recorded at a distance of 0.3 mm from the stimulating electrode

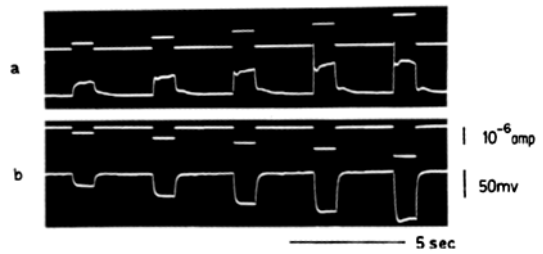

FIGURE 9. Electrotonic potentials and spikes of the longitudinal muscle of the antrum evoked by the extracellular polarizing method. a, depolarizing currents under various intensities. b, hyperpolarizing currents under various intensities.

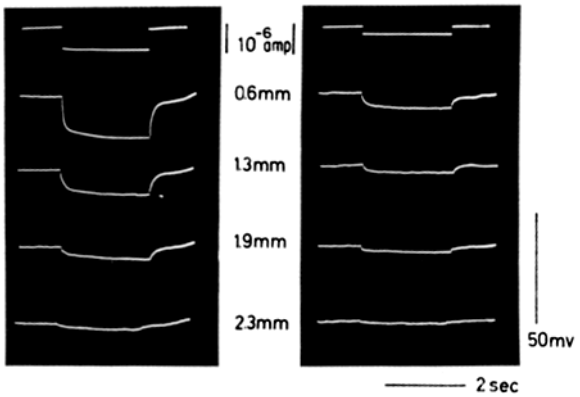

FIGURE 10. Electrotonic potentials of the longitudinal muscle of the antrum recorded at four different distances from the stimulating electrode. Two different intensities of hyperpolarizing current were used.

(i.e. 0.25 λ) with the application of a weak cathodal pulse (1 sec). Fig. 9 shows the electrotonic potentials and spikes of the longitudinal muscle of the antrum evoked by extracellularly applied depolarizing (a) and hyperpolarizing currents (b). The recording electrode was placed at a distance of 0.2 mm from the stimulating electrode. Then current-voltage relations were measured by application of the polarizing currents. The longitudinal and circular muscles of the antrum showed remarkable rectification. This membrane rectification observed by the extracellular polarizing method was more marked than that observed by the intracellular polarizing method.

The space constants (λ) of the longitudinal and circular muscles of the antrum were measured by the extracellular polarizing method. The amplitudes of the electrotonic potential decreased exponentially with distance from the stimulating electrode. Fig. 10 shows the electrotonic potentials of the longitudinal muscle of the antrum determined at four different distances from the stimulating electrode. Two different intensities of hyperpolarizing currents were used. Fig. 11 shows the relation between the amplitude of the electrotonic potential and the distance from the stimulating electrode. The space constant calculated in this way was 1.1 mm (n = 3) for the longitudinal muscle and 1.4 mm (n = 3) for the antrum circular muscle.

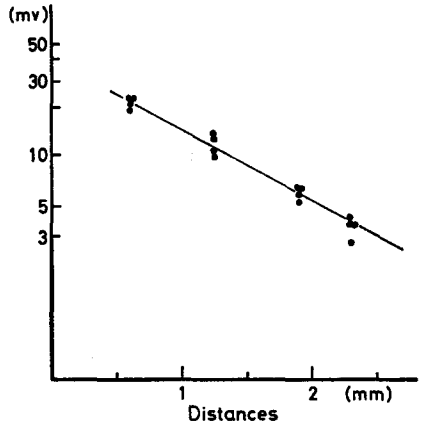

FIGURE 11. Relationship between the amplitudes of the electrotonic potential recorded from the longitudinal muscle and distances of the potential recording site from the stimulating electrode. The amplitudes of the electrotonic potential were scaled semilogarithmically.

These observations strongly indicate that the longitudinal and circular muscles of the antrum show cable-like properties for the propagation of excitation as a unit (or functional bundle).

DISCUSSION

Differences between intestinal and stomach muscle are apparent from the characteristics of the spontaneous discharges of spikes, slow waves, and the membrane properties observed by the extracellular stimulation. The slow depolarization of the membrane in the stomach is probably not due to the same mechanism as the plateau potential of the cardiac muscle (Weidmann, 1956) or the slow plateau potential of the guinea pig ureter (Bennett, Burnstock, Holman, and Walker, 1962; Washizu, 1966; Kuriyama, Osa, and Toida, 1967 *a*), because, in the stomach, the spike discharge appeared on a plateau phase and the slow depolarization behaved like a generator potential. In cardiac muscle, the spike potential is never superimposed on the plateau phase. In the guinea pig ureter, the slow plateau potential never preceded the generation of the spike, and the first spike was followed by the generation of the plateau. The long duration and relatively high amplitude of the slow

wave appear to be characteristic of the stomach which differs from other parts of the alimentary canal. The number of spikes which appear superimposed on the slow depolarization is smaller in the fundus, i.e. greater curvature of the antrum, than in the pylorus. This might be related to the strong phasic contractions which occur in the pyloric region.

The spike could be elicited by electrical stimulation in the presence of tetrodotoxin but it was blocked by Mn^{++}. The spike could also be elicited in the sodium-free sucrose solution. These effects were the same as those observed in the intestine (Nonomura, Hotta, and Ohashi, 1966; Kuriyama, Osa, and Toida, 1966, 1967 *c*). However, in the presence of tetrodotoxin the slow potential was reduced in amplitude and finally ceased, thereby causing block of spontaneous spike generation. Atropine also blocked the generation of the slow potential and the spike in three out of five specimens. In guinea pig intestine, on the other hand, spontaneous spike generation continues unchanged in the presence of tetrodotoxin and atropine. These observations indicate that the mechanism of spontaneous spike generation, related to the mechanism of automaticity, may be different in the stomach than in intestinal smooth muscle. The slow wave not only ceased after treatment with tetrodotoxin and atropine, but also it was enhanced in amplitude by prostigmine. These observations might indicate that the cholinergic nerve fibers and plexus distributed between the muscle layers play an important role in the generation of the slow wave. However, in comparison to intestinal smooth muscle the stomach muscle, and especially its slow wave response, showed very little sensitivity to bulk application of acetylcholine. Presumably low sensitivity to acetylcholine and the marked effect of prostigmine might be due to high acetylcholine–esterase activity in the stomach. These different effects of acetylcholine on the intestine and stomach can only be solved by further investigation. Furthermore, the nature of the slow waves which have less sensitivity to cholinergic substances must be further clarified.

Papasova, Nagai, and Prosser (1968) described extracellular records from cat stomach which showed two components of the slow wave, i.e. the initial rapid component which was sodium-dependent, was propagated, and was not correlated with contraction, and a second component which was calcium-dependent, was abolished by Mn^{++}, was not propagated but was always found when contractions occurred. These two components of the slow wave have been previously postulated from the dog stomach by Daniel (1965). In the present experiments, we could not distinguish two components of the slow wave when recording from single cells of the guinea pig stomach.

The chronaxie for the spike generation of the longitudinal muscle of the antrum of guinea pig had a value (26 msec) similar to that for other parts of the intestine (taenia coli 20–30 msec [Bülbring and Tomita, 1967], jejunum 25 msec, rectum 5–18 msec [Kuriyama et al., 1967 *b*]). Conduction velocity

of excitation measured in the longitudinal muscle of the antrum was 1.2 cm/sec and this value is lower than that measured in the intestine (taenia coli 6.7–8.8 cm/sec [Bülbring, Burnstock, and Holman, 1958], 7.3 cm/sec [Tomita, 1966 *a*, *b*], jejunum 3.2 cm/sec [Kuriyama et al., 1967 *b*], rectum, 4.8 cm/sec [Kuriyama et al., 1967 *b*]). The conduction velocity measured from the dog stomach muscle was 6–12 mm/sec (Richter, 1923), 1.5–3.0 mm/sec (Bozler, 1945), and 4–11 mm/sec in the cat (Papasova, Nagai, and Prosser, 1968). These values for the conduction velocity are nearly a tenth of those measured from the intestine. The mechanism for the low conduction velocity in the stomach is discussed elsewhere by Osa (data to be published).

Shuba (1961) measured the electrotonus in the frog stomach smooth muscle cell using extracellularly applied currents and he reported that the rise time of the slow part of the electrotonic potential is much slower than for the corresponding part in the nerve and striated muscle. He concluded that the spread of electrotonus over quite a long distance from the site of its origin suggests the presence of a certain continuous morphological link between the muscle cells.

The results of the present experiments also supported the view that the longitudinal and circular muscle tissues show cable properties as a functional unit but not as a single cell. Intracellular polarizing current applied by the Wheatstone bridge method spread to the neighboring cells and consequently the induced potential changes might not indicate the membrane property of the single cell. On the other hand, as expected from the cable properties, extracellularly applied currents evoked the electrotonic potentials.

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