

Myogenic peristalsis in isolated preparations of chicken oesophagus

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Summary

1. Nicotine, 20 $\mu\text{g/ml}$, briefly and partially blocked the longitudinal contractions of a chick vagus nerve–oesophagus preparation to nerve stimulation, but potentiated them in the presence of hyoscine, 10 $\mu\text{g/ml}$.
2. Strychnine, 20 $\mu\text{g/ml}$, antagonized the longitudinal contractions of the chick oesophagus to nerve stimulation, though not to acetylcholine or 5-hydroxytryptamine. Hyoscine, 10 $\mu\text{g/ml}$, enhanced the nerve-blocking action of strychnine.
3. These observations suggest that in the vagus nerve of the chicken, hyoscine either blocks certain types of fibre selectively or interacts with pharmacological receptors for strychnine and nicotine.
4. Tetrodotoxin, 1 $\mu\text{g/ml}$, propranolol, 40 $\mu\text{g/ml}$, cocaine, 200 $\mu\text{g/ml}$, or strychnine, 100 $\mu\text{g/ml}$, which render autonomic nerves inexcitable, potentiated the spontaneous movements of isolated preparations of chicken oesophagus at normal intra-luminal pressure; they also potentiated peristaltic response to increased intra-luminal pressure.
5. These observations suggest that the preparations contain intramural nerves that inhibit the musculature.
6. Peristalsis in the chicken oesophagus was unaffected by nicotine, 20 $\mu\text{g/ml}$, tubocurarine, 20 $\mu\text{g/ml}$, hyoscine, 10 $\mu\text{g/ml}$, phentolamine, 1 $\mu\text{g/ml}$, propranolol, 0.5 $\mu\text{g/ml}$, mepyramine, 1 $\mu\text{g/ml}$, methysergide, 0.1 $\mu\text{g/ml}$, and morphine, 20 $\mu\text{g/ml}$. It was produced in preparations of chicken oesophagus after removal of the mucous membrane. Papaverine, 20 $\mu\text{g/ml}$, inhibited the peristalsis and had a spasmolytic action in the musculature.
7. These observations suggest that the peristalsis in isolated preparations of chicken oesophagus is dependent solely upon myogenic contractility.

Introduction

The nerve plexuses in the chicken oesophagus are situated between the longitudinal and circular muscle in the externa and in close apposition to the muscularis mucosae; the musculature consists solely of plain muscle (Bartlet & Hassan, 1968a; Calhoun, 1933; 1954). Stimulation of the extrinsic parasympathetic nerves produces contractions of the muscles in the pre-crop oesophagus which are only partly antagonized by tubocurarine, hexamethonium and hyoscine (Bartlet, 1972; Hassan, 1969). In the present experiments with isolated preparations of chicken oesophagus, the reflex produced by an increased intra-luminal pressure (peristaltic) was recorded by a modification of the method described by Trendelenburg (1917). Some of the

drugs used in the pharmacological analysis of the reflex were tested on a vagus nerve-oesophagus preparation to ascertain whether they blocked nerves in the preparations.

Methods

Vagus nerve-oesophagus and Remak nerve-rectum preparations

Vagus nerve-oesophagus and Remak nerve-rectum preparations were made from chicks aged 1-13 days as described by Bartlet & Hassan (1968a, 1971). The preparations were mounted in a 50 ml organ bath filled with Krebs solution gassed with 5% CO₂ in oxygen and maintained at 35° C. The nerves were stimulated through platinum electrodes with square wave pulses (1 ms, 25 Hz, 2-10 V) which were monitored. The vagus nerve was stimulated for 5 s or 10 s in the presence of hyoscine and the Remak nerve 10 s, with an interval of 10 or 15 min between trains of stimuli. The responses were recorded on smoked paper with an isotonic lever, load 1.5 g, which magnified the responses 10 or 20 times.

Preparations of oesophagus for recording peristalsis

Pullets, young cockerels and hens, body weights ranging from 0.9 to 2.5 kg, were killed with an air embolus. Two segments of the oesophagus were rapidly dissected and put into Krebs solution; the pre-crop preparation comprised a 6-7 cm length of the organ taken from just above the crop and the post-crop preparation the segment between the crop and the proventriculus. Preparations of the post-crop oesophagus were made from 33 chickens and preparations of both pre- and post-crop oesophagus were obtained from a further 17 birds. The mucous membrane was removed from four preparations of post-crop oesophagus. A thread attached to the oral end of the segment was gently pulled through the lumen of the preparation to evert it. The oesophagus was then drawn on to a glass rod (diameter 9 mm) and the mucous membrane incised longitudinally and peeled away. The preparation was returned to its normal position for the experiment.

The caudal end of the preparations was tied to a J-tube (internal diameter 4 mm) connected to a reservoir containing 0.9% w/v NaCl in water. After washing the lumen of the preparation with saline a thread was tied around its oral end so that the intra-luminal pressure could be controlled by adjusting the reservoir height. The reservoir was sealed with a ground glass stopper and the pressure above the saline monitored with a Greer differential micromanometer. The output from the micromanometer was put through a variable resistance to a pen driver amplifier of a Devices 4-channel recorder, adjustment of the resistance setting the sensitivity. The micromanometer was calibrated and produced a linear record of changes in intra-oesophageal volume. The oral end of the preparation was attached to an isotonic displacement meter against a load of 2.5 g. The output from the isotonic displacement meter was fed through a pre-amplifier (type DC.2C) to a second pen driver amplifier in the recorder. The isotonic displacement meter was calibrated and produced a linear record of the longitudinal contractions of the preparation.

The preparations were mounted in a 75 ml organ bath filled with Krebs solution gassed with 5% CO₂ in oxygen and maintained at 37° C and were rested for half an hour before the commencement of an experiment. Preliminary experiments showed that an increase in intra-luminal pressure of 10 to 15 cm H₂O was just

sufficient to initiate propulsive activity in the post-crop preparation. In the experiments to be described, the intra-luminal pressure was increased by 15 cm H₂O for periods of 1 min at intervals of 20 minutes.

Chemicals

A bathing solution of the composition described by Krebs & Henseleit (1932) was made with ANALAR salts and de-ionized water. Drugs were added to the Krebs solution which bathed the outer surface of the preparations for a period of 18 or 38 min, unless otherwise indicated under **Results**. The drugs used were cocaine hydrochloride B.P. (MacFarlane Smith), (-)-hyoscine hydrobromide (B.D.H.), mepyramine maleate (May & Baker), methysergide bimaleate (Sandoz), morphine sulphate B.P. (MacFarlane Smith), nicotine hydrogen tartrate (B.D.H.), papaverine hydrochloride B.P. (Evans Medical Supplies), phentolamine mesylate (Ciba), picrotoxin (Sigma), (\pm)-propranolol hydrochloride (I.C.I.), strychnine hydrochloride (B.D.H.), tetrodotoxin (Sigma) and (+)-tubocurarine hydrochloride B.P. (Burroughs Wellcome). The drug concentrations in the text, table and figures are concentrations of these compounds in the organ bath.

Results

Effects of nicotine, strychnine and papaverine on responses of the longitudinal muscle in the oesophagus and rectum to extrinsic nerve stimulation and drugs

Nicotine, 20 μ g/ml, produced a strong contraction in the oesophagus followed by a transient and incomplete blockade of the response to stimulation of a vagus nerve (Fig. 1). Five min after the addition of nicotine to the bathing solution, the height of the contraction to nerve stimulation was reduced by $52.0 \pm 13.4\%$ ($n=4$, $t_{calc}=3.9$, $P<0.02$), but the response soon recovered despite the continued presence of the drug.

Since nerve stimulation produces both hyoscine-sensitive and hyoscine-insensitive contractions in the chicken oesophagus (Bartlet, 1972; Hassan, 1969), the effect of nicotine was also examined in preparations contracting to nerve stimulation in the

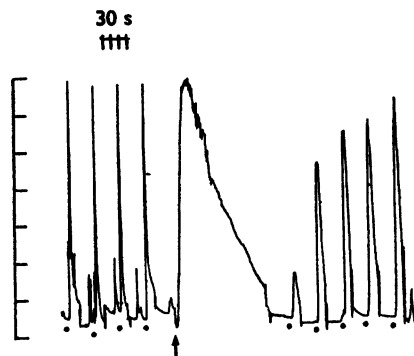


FIG. 1. Vagus nerve-oesophagus preparation. The dots mark 5 s periods of nerve stimulation (1 ms, 25 Hz, 2 V) at 10 min intervals. From the arrow to the end of the tracing nicotine, 20 μ g/ml, was present. Nicotine produced a transient and incomplete blockade of the response to nerve stimulation. Vertical scale, cm. Magnification $\times 10$. Time, 30 s.

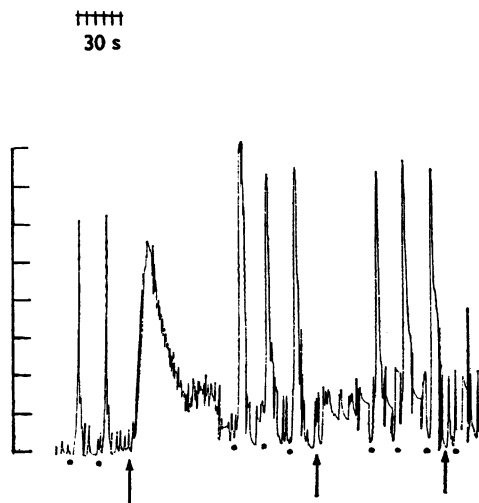


FIG. 2. Vagus nerve-oesophagus preparation with hyoscine, 10 $\mu\text{g}/\text{ml}$, in the bathing solution. The dots mark 10 s periods of nerve stimulation (1 ms, 25 Hz, 5 V) at 10 min intervals. The arrows mark the addition of nicotine, 20 $\mu\text{g}/\text{ml}$, nicotine, 100 $\mu\text{g}/\text{ml}$, and tetrodotoxin, 0.1 $\mu\text{g}/\text{ml}$, respectively, to the bathing solution. The drugs were not washed out of the organ bath. In the presence of hyoscine, nicotine potentiated the response to nerve stimulation. Vertical scale, cm. Magnification $\times 10$. Time, 30 s.

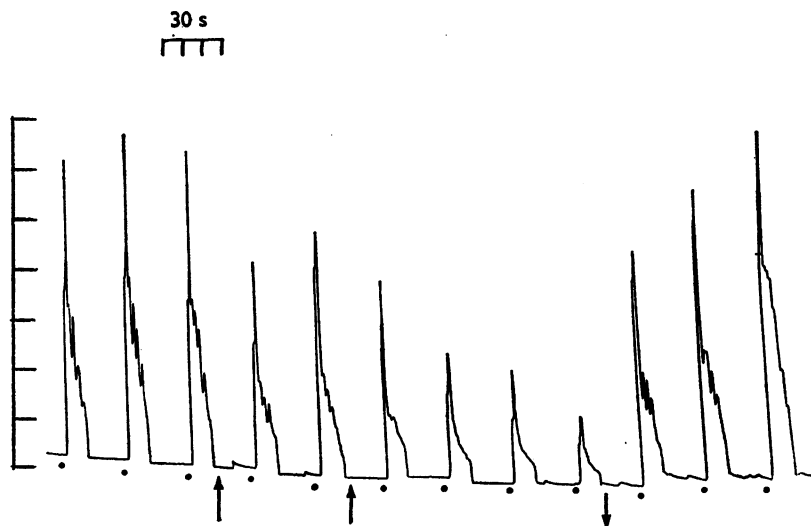


FIG. 3. Vagus nerve-oesophagus preparation with hyoscine, 10 $\mu\text{g}/\text{ml}$, in the bathing solution. The dots mark 10 s periods of nerve stimulation (1 ms, 25 Hz, 5 V) at 15 min intervals. The first two arrows mark additions of strychnine at concentrations of 2.5 and 5 $\mu\text{g}/\text{ml}$, respectively; at the third arrow strychnine was washed out of the organ bath. Strychnine antagonized the contractions to nerve stimulation, the antagonism being readily reversed. Vertical scale, cm. Magnification $\times 10$. Time, 30 s.

presence of hyoscine, 10 $\mu\text{g}/\text{ml}$. In these preparations, nicotine, 20 $\mu\text{g}/\text{ml}$, produced a marked contraction followed by a transient potentiation of the response to nerve stimulation (Fig. 2). Five min after exposure to nicotine the height of the contraction to nerve stimulation was increased by $49.6 \pm 17.2\%$ ($n=4$, $t_{\text{calc}}=2.8$, $P<0.05$). In the experiment shown in Fig. 2 the response to nerve stimulation in the presence of nicotine, 20 $\mu\text{g}/\text{ml}$, was unaffected by nicotine at a higher concen-

TABLE 1. Antagonism by strychnine of responses of the oesophagus to vagal stimulation

Concentration ($\mu\text{g/ml}$)	Percentage inhibition of response to stimulation for	
	5 s	10 s in presence of hyoscine (10 $\mu\text{g/ml}$)
5		62 \pm 9 (5)
10	30 \pm 6 (6)	82 \pm 9 (5)

The number of preparations are given in parentheses. They were stimulated with 1 ms pulses at 25 Hz, and the antagonism was measured after 1 h exposure to strychnine.

tration and was abolished by tetrodotoxin, 0.1 $\mu\text{g/ml}$. In 3 of the 5 experiments the potentiation of the response to nerve stimulation in the presence of nicotine was not maintained.

Nicotine, 20 $\mu\text{g/ml}$, abolished contractions in 5 preparations of chicken rectum produced by stimulation of Remak's nerve. In all the experiments, propranolol, 0.05 $\mu\text{g/ml}$, and phentolamine, 0.1 $\mu\text{g/ml}$, were included in the bathing solution to antagonize the relaxation of the preparation to nerve stimulation (Bartlet & Hassan, 1971), and in 2 of the experiments hyoscine, 10 $\mu\text{g/ml}$, was also present.

Strychnine antagonized the contractions of the oesophagus produced by nerve stimulation, the antagonism being readily reversed on washing strychnine out of the organ bath (Fig. 3). The potency of the nerve-blocking action of strychnine was increased by hyoscine, 10 $\mu\text{g/ml}$ (Table 1). Contractions of the oesophagus produced by acetylcholine and 5-hydroxytryptamine were not antagonized by strychnine, 20 $\mu\text{g/ml}$, although exposure to a very high concentration (100 $\mu\text{g/ml}$) for 1 h produced a slight inhibition of the responses to these drugs.

Exposure of the preparations to papaverine, 10 $\mu\text{g/ml}$, blocked the longitudinal contractions produced by nerve stimulation, acetylcholine and 5-hydroxytryptamine, but morphine, 10 $\mu\text{g/ml}$, did not affect the responses of the oesophagus to these stimulants (3 experiments in each instance).

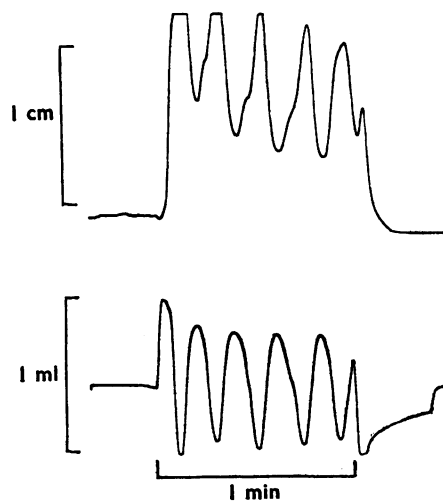


FIG. 4. Preparation of pre-crop oesophagus. From above downwards: longitudinal contractions (cm) and changes in intra-luminal volume (ml). The intra-luminal pressure was increased by 15 cm H_2O for the period marked by the horizontal bracket.

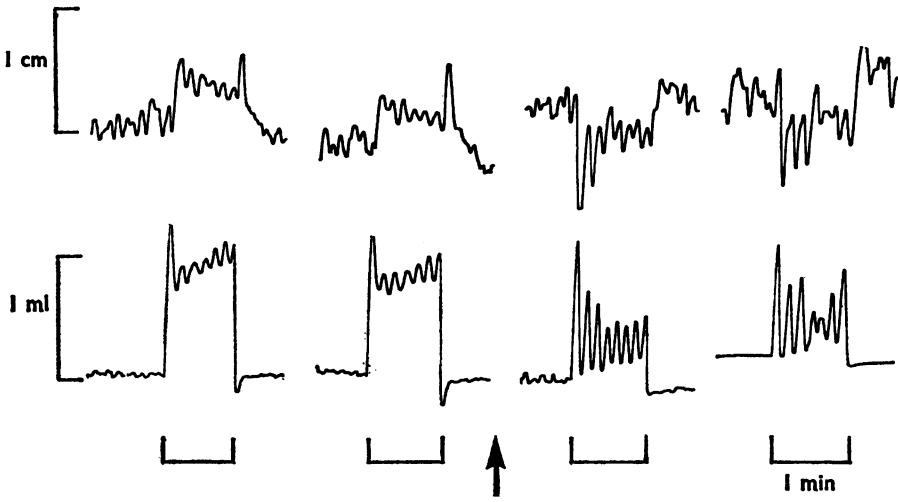


FIG. 5. Preparation of post-crop oesophagus. From above downwards: longitudinal contractions (cm) and changes in intra-luminal volume (ml). Each horizontal bracket marks a one minute period during which the intra-luminal pressure was increased by 15 cm H₂O. From the arrow to the end of the trace tetrodotoxin, 1 μ g/ml, was present. Tetrodotoxin potentiated the peristalsis. Eighteen min elapsed between each section of the trace.

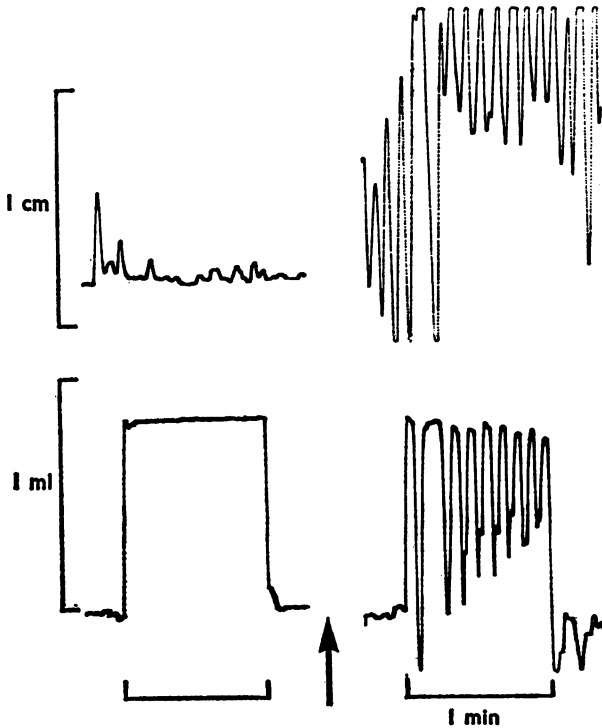


FIG. 6. Preparation of pre-crop oesophagus. From above downwards: longitudinal contractions (cm) and changes in intra-luminal volume (ml). The horizontal brackets mark one minute periods during which the intra-luminal pressure was increased by 15 cm H₂O. From the arrow to the end of the trace cocaine, 200 μ g/ml, was present. Cocaine potentiated peristalsis. Eighteen min elapsed between the sections of the trace.

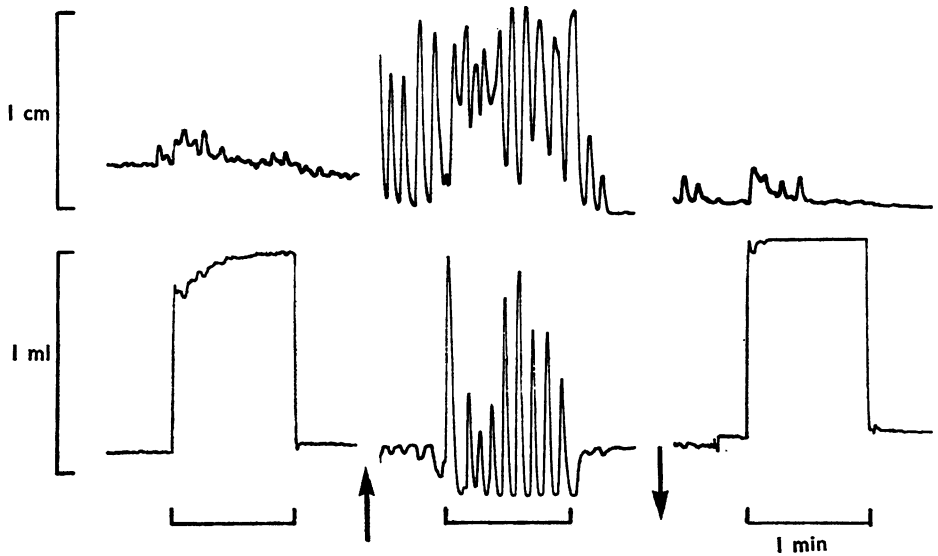


FIG. 7. Preparation of pre-crop oesophagus. From above downwards: longitudinal contractions (cm) and changes in intra-luminal volume (ml). The horizontal brackets mark one minute periods during which intra-luminal pressure was increased by 15 cm H₂O. Between the arrows strychnine, 100 µg/ml, was present in the bathing solution. Strychnine facilitated peristalsis. Eighteen min elapsed between the sections of the trace.

Response of the oesophagus produced by an increase in intra-luminal pressure

The preparations of pre-crop oesophagus usually remained quiescent when the intra-luminal pressure was increased by 3–25 cm H₂O (see Figs. 6 and 7). In two pre-crop preparations, propulsive contractions were produced when the intra-luminal pressure was increased by 15 cm H₂O, each propulsive contraction commencing when a longitudinal contraction had partly developed (Fig. 4).

The post-crop preparations exhibited spontaneous activity which consisted of small longitudinal contractions (see Figs. 5, 8 and 10). Neither an increase in intra-luminal pressure nor the ensuing propulsive contractions were associated with any consistent change in the length of the preparation. In most of the post-crop preparations an increase in intra-luminal pressure of 15 cm H₂O produced propulsive activity which was seen to be the outcome of circular contractions which were localized or propagated through a part of the preparation only. Five to ten circular contractions were produced when the intra-luminal pressure was increased for 1 min, the first contraction was frequently larger than the rest.

The preparations which did not show the peristaltic reflex initially did so after exposure to a drug which facilitated peristalsis in chicken oesophagus.

The effect of drugs which render nerves inexcitable on the response to an increase in intra-luminal pressure

In two preparations of post-crop oesophagus, exposure to tetrodotoxin, 1 µg/ml, potentiated the propulsive activity produced by an increase in intra-luminal pressure (Fig. 5). In two further experiments, both the inner and outer surfaces of the post-crop oesophagus were bathed with tetrodotoxin, 1 µg/ml, and hyoscine, 10 µg/ml; these preparations still exhibited a strong peristaltic reflex. In the presence

of tetrodotoxin, the circular contractions were propagated and sometimes expelled a volume of saline which exceeded the distension arising from the increase in intra-luminal pressure.

Cocaine, 200 $\mu\text{g}/\text{ml}$, and propranolol, 40 $\mu\text{g}/\text{ml}$, both potentiated peristalsis in the chicken oesophagus. Cocaine was tested in 4 preparations each of the pre- and post-crop oesophagus and propranolol in 2 pre-crop preparations and 3 post-crop preparations. The effect of these drugs was especially striking in the preparations of pre-crop oesophagus since peristalsis was usually absent before exposure to the drug (Fig. 6). In one preparation of pre-crop oesophagus exposed to cocaine, 200 $\mu\text{g}/\text{ml}$, an increase in intra-luminal pressure produced circular contractions which were propagated orally. These contractions were not recorded since they produced distension of the oral end of the segment and did not propel the intra-luminal contents into the J-tube.

Strychnine facilitated the peristaltic reflex in the chicken oesophagus. Exposure to strychnine, 100 $\mu\text{g}/\text{ml}$, for 18 min produced strong peristalsis in 2 preparations of pre-crop oesophagus (Fig. 7) and 4 post-crop preparations, the effect of the drug being readily reversed upon washing the preparation. Strychnine, 20 $\mu\text{g}/\text{ml}$, potentiated peristalsis in 4 preparations of post-crop oesophagus, but in two of the preparations the propulsive activity was weak after 38 min exposure to the drug. Exposure to strychnine, 4 $\mu\text{g}/\text{ml}$, for 38 min did not potentiate peristalsis in 2 preparations of post-crop oesophagus. Picrotoxin, 500 $\mu\text{g}/\text{ml}$, did not affect peristalsis in the oesophagus (2 preparations each of pre- and post-crop oesophagus).

Effect of antagonists of acetylcholine and noradrenaline on the response to an increase in intra-luminal pressure

The propulsive activity in the oesophagus produced by an increase in intra-luminal pressure was not affected on exposure to tubocurarine, 20 $\mu\text{g}/\text{ml}$ (2 post-crop preparations). Exposure to nicotine, 20 $\mu\text{g}/\text{ml}$, produced a strong longitudinal contraction and a small decrease in intra-luminal volume but it did not facilitate peristalsis in 2 preparations of post-crop oesophagus and 3 pre-crop preparations

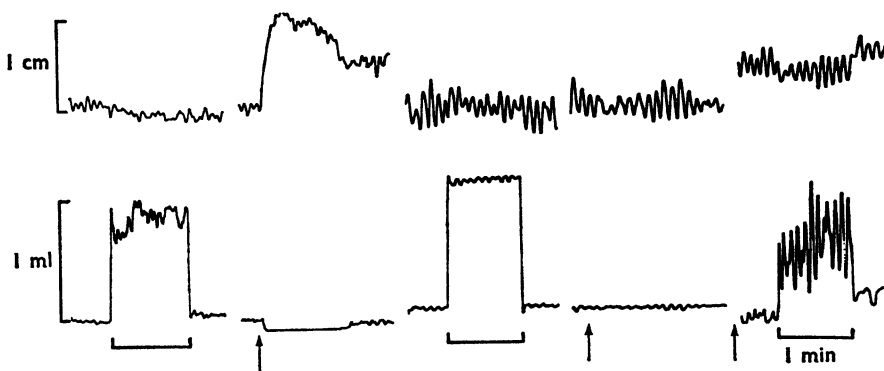


FIG. 8. Preparation of post-crop oesophagus. From above downwards: longitudinal contractions (cm) and changes in intra-luminal volume (ml). The horizontal brackets mark one minute periods during which the intra-luminal pressure was increased by 15 cm H.O. From the first arrow to the end of the trace nicotine, 20 $\mu\text{g}/\text{ml}$, was present in the bathing solution. The second and third arrows mark the addition of nicotine, 100 $\mu\text{g}/\text{ml}$, and propranolol, 40 $\mu\text{g}/\text{ml}$, respectively, to the organ bath. The peristalsis was unaffected by nicotine and potentiated by propranolol. Eighteen min elapsed between the sections of the trace.

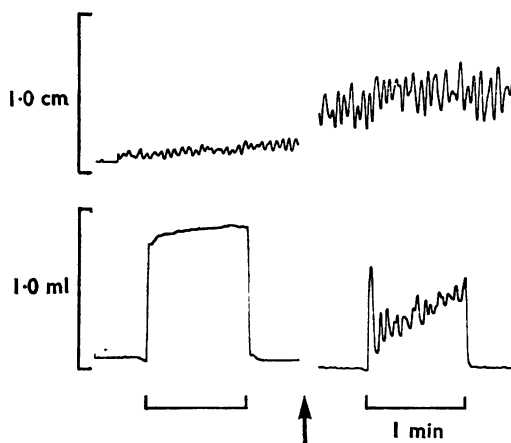


FIG. 9. Preparation of post-crop oesophagus with mucous membrane removed. From above downwards: longitudinal contractions (cm) and changes in intra-luminal volume (ml). The horizontal brackets mark min when the intra-luminal pressure was increased by 15 cm H₂O. From the arrow to the end of the trace cocaine, 200 μ g/ml, was present. Cocaine facilitated peristalsis. Eighteen min elapsed between the sections of the trace.

(Fig. 8). The response of the musculature to nicotine indicated that the intramural nerves were functional since nicotine did not produce contractions in 2 preparations of chicken oesophagus in which the nerves had been rendered inexcitable with tetrodotoxin, 0.1 μ g/ml.

Exposure of the post-crop preparation to hyoscine, 10 μ g/ml, did not modify the response to an increase in intra-luminal pressure (5 experiments). Hyoscine, 10 μ g/ml, was also tested on 2 preparations of pre-crop oesophagus in which propulsive activity was not produced upon increasing the intra-luminal pressure either in the absence or presence of the drug. Hyoscine, in one ten-thousandth of the concentration used in the present experiments, antagonized acetylcholine in the chicken oesophagus (Bartlet & Hassan, 1968b).

The response of the isolated oesophagus to an increase in intra-luminal pressure was unaffected on exposure to a combination of phentolamine, 1 μ g/ml, and propranolol, 0.5 μ g/ml (2 preparations of pre-crop oesophagus and 2 post-crop preparations). A tenth of these concentrations of phentolamine and propranolol is sufficient to block adrenoceptors in the chick rectum preparation (Bartlet & Hassan, 1970).

At the end of the antagonism experiments the peristaltic reflex was tested after exposure of the preparation to cocaine, 200 μ g/ml, or propranolol, 40 μ g/ml. Each preparation exhibited strong peristalsis in the presence of one of these drugs (see Fig. 8).

The effect of removing the mucous membrane

Preparations of post-crop oesophagus with the mucous membrane removed did not develop propulsive activity when the intra-luminal pressure was increased; however, 3 of the 4 preparations exhibited some peristalsis after exposure to cocaine, 200 μ g/ml (Fig. 9). Two of the preparations, which were embedded in paraffin wax and examined histologically at the end of the experiments, showed that the mucosa had been divided at the level of the muscularis mucosae.

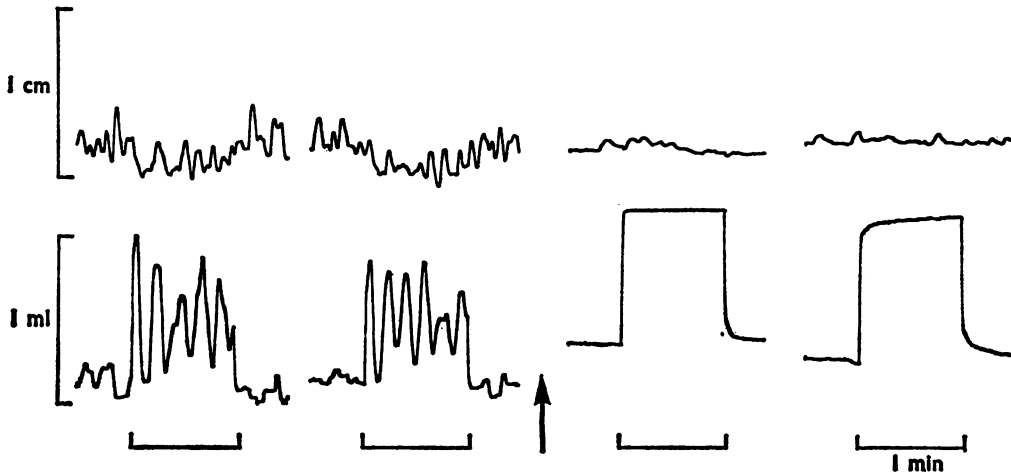


FIG. 10. Preparation of post-crop oesophagus. From above downwards: longitudinal contractions (cm) and changes in intra-luminal volume (ml). The horizontal brackets mark one minute periods during which intra-luminal pressure was increased by 15 cm H₂O. From the arrow to the end of the trace papaverine, 20 μ g/ml, was present. Papaverine inhibited peristalsis. Eighteen minutes elapsed between the sections of the trace.

The effect of papaverine, morphine, mepyramine and methysergide on the response to an increase in intra-luminal pressure

The propulsive activity in preparations of post-crop oesophagus produced by an increase in intra-luminal pressure was abolished by papaverine, 20 μ g/ml (3 preparations, Fig. 10), and was not affected by morphine, 20 μ g/ml (2 preparations), mepyramine, 1 μ g/ml (3 preparations) and methysergide, 0.1 μ g/ml (3 preparations). These concentrations of mepyramine and methysergide were sufficient to antagonize the actions of histamine and 5-hydroxytryptamine, respectively, in the chicken oesophagus preparation (Bartlet & Hassan, 1968b).

Discussion

Although tubocurarine blocks ganglionic transmission in the Remak nerve-rectum preparation it has very little effect on the contractions of the chicken oesophagus produced by stimulation of a vagus nerve (Bartlet & Hassan, 1971; Hassan, 1969). Similarly, in the present experiments, nicotine abolished the contractions of the rectum to nerve stimulation but not those of the oesophagus. Nicotine only produced a transient and incomplete blockade of the oesophageal contractions to nerve stimulation, and even this effect was not apparent when hyoscine was present in the bathing solution. This modification of the response to nicotine suggests a pharmacological dissimilarity in ganglionic transmission in the absence and presence of hyoscine. Strychnine, 20 μ g/ml, antagonized the response of the chicken oesophagus to nerve stimulation though not to acetylcholine and 5-hydroxytryptamine, which act directly on the muscle (Bartlet & Hassan, 1968a & b). Thus the inhibitory action of strychnine was in the peripheral nerve. The potency of strychnine as a nerve-blocking agent was enhanced by hyoscine. These observations suggest that in the vagus nerve of the chicken, hyoscine either blocks certain types of fibre selectively or interacts with pharmacological receptors for strychnine and nicotine.

As expected, tubocurarine and nicotine did not affect the peristaltic reflex in the chicken oesophagus, since these drugs have been shown to be ineffective in blocking ganglionic transmission in the preparation. Tetrodotoxin, 1 $\mu\text{g/ml}$, and large concentrations of propranolol (40 $\mu\text{g/ml}$) and cocaine (200 $\mu\text{g/ml}$) render the autonomic nerves in isolated organs inexcitable (Bartlet & Hassan, 1968a, 1969; Gershon, 1967). Exposure of the preparations to one of these drugs or to strychnine, 20 $\mu\text{g/ml}$ and above, facilitated the peristaltic reflex, which suggests that the chicken oesophagus contains nerves which may inhibit peristalsis. However, blockade of the neural structures with drugs usually resulted in an increase in the spontaneous movements of the preparations in addition to a facilitation of peristalsis. Thus the musculature seemed to be inhibited by neural structures when the intra-luminal pressure was at the normal level. In some preparations a strong peristaltic reflex was demonstrable without exposure to a nerve-blocking drug; thus an increase in intra-luminal pressure did not seem to stimulate the inhibitory nerves. Antagonists of acetylcholine and noradrenaline, in concentrations usually effective, did not affect peristalsis in the chicken oesophagus preparation, so that the inhibitory nerves did not seem to be cholinergic or adrenergic.

The propagated waves of peristalsis seemed to be independent of intrinsic neural pathways since they were produced when nervous transmission had been blocked by drugs and after removal of the mucous membrane. Stretch has a depolarizing effect in plain muscle, which may be sufficient to produce a discharge of spikes (Gillespie, 1962). Thus plain muscle opposes a stretching force by contraction. This property of myogenic contractility seemed to be the basis of the peristalsis observed in the present experiments. In one preparation the peristalsis was propagated orally. This observation suggests that plain muscle cells in the caudal end of the preparations may comprise the pacemaker when the intra-luminal pressure is increased throughout the segment. Swallowing would produce a pacemaker at the oral end of the oesophagus and peristalsis propagated caudally. The peristalsis in the preparations from which the mucous membrane had been removed was weaker than in full thickness preparations. Removal of the mucous membrane may have impaired peristalsis through damage to the nexuses or some other structure in the muscle (Barr, Berger & Dewey, 1968). The peristalsis observed in the present experiments was unlike that produced in the intestines of several mammalian species, which is mediated by an intrinsic reflex arc consisting of sensory and motor neurones (Bülbring, Lin & Schofield, 1958).

Papaverine inhibited peristalsis and the longitudinal contractions produced by nerve stimulation, acetylcholine and 5-hydroxytryptamine, demonstrating that it has a direct inhibitory action in the plain muscle of the chicken oesophagus. Mepyramine and methysergide did not affect the response of the preparations to an increase in intra-luminal pressure, indicating that the actions of histamine and 5-hydroxytryptamine, respectively, are not an integral part of the peristaltic reflex in the chicken oesophagus.

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