Acta Medica Okayama

Volume 44, *Issue* 2 1990 *Article* 8

APRIL 1990

Regulation of bile duct motility by vagus and sympathetic nerves in the pigeon.

Toshiaki Neya[*] Teruhiro Yamasato[†]

Masatoshi Mizutani[‡] Sosogu Nakayama[**]

[*]Okayama University,

[†]Okayama University,

[‡]Okayama University,

[**]Okayama University,

Regulation of bile duct motility by vagus and sympathetic nerves in the pigeon.*

Toshiaki Neya, Teruhiro Yamasato, Masatoshi Mizutani, and Sosogu Nakayama

Abstract

Effects of stimulation of the vagus and sympathetic nerves on bile duct peristalses were studied in pigeons anesthetized with urethane. Vagus stimulation increased the frequency of peristalses. Atropine, hexamethonium and tetrodotoxin abolished this excitatory effect. After atropine, inhibition of peristalses sensitive to tetrodotoxin was produced. Stimulation of sympathetic area in the spinal cord inhibited peristalses. Propranolol converted this effect into an excitatory one, which was abolished by phentolamine. The results suggest that vagal and sympathetic innervations of the bile duct in pigeons are similar to those of the sphincter of Oddi in mammalian species.

KEYWORDS: biliary system, bile duct, motility, innervation, pigeon

---

*PMID: 2363364 [PubMed - indexed for MEDLINE]

Acta Med Okayama 44 (2) 113-115 (1990)

— Brief Note —

Regulation of Bile Duct Motility by Vagus and Sympathetic Nerves in the Pigeon

Toshiaki Neya*, Teruhiro Yamasato, Masatoshi Mizutani and Sosogu Nakayama

Department of Physiology, Okayama University Medical School, Okayama 700, Japan

Effects of stimulation of the vagus and sympathetic nerves on bile duct peristalses were studied in pigeons anesthetized with urethane. Vagus stimulation increased the frequency of peristalses. Atropine, hexamethonium and tetrodotoxin abolished this excitatory effect. After atropine, inhibition of peristalses sensitive to tetrodotoxin was produced. Stimulation of sympathetic area in the spinal cord inhibited peristalses. Propranolol converted this effect into an excitatory one, which was abolished by phentolamine. The results suggest that vagal and sympathetic innervations of the bile duct in pigeons are similar to those of the sphincter of Oddi in mammalian species.

Key words : biliary system, bile duct, motility, innervation, pigeon

In most mammalian species, the common bile duct has only sparse outer muscle layers, but the sphincter of Oddi, a terminal portion of the common bile duct, has well-developed muscles (1). In the bird, the bile duct has well-developed outer muscle layers, but there is no distinct sphincter structure (2). It is understood that bile evacuation into the duodenum is regulated by the motor activity of the sphincter of Oddi in the former (3) and by peristalsis of the duct in the latter (2, 4, 5). The sphincter of Oddi is embedded in the duodenal wall and its motility can be influenced passively by the duodenal response to extrinsic nerve stimulation. Thus, to evaluate the results of extrinsic nerve actions on the sphincter of Oddi in mammals reported previously (6-9), it is available to reveal the action of extrinsic nerves on the pigeon bile duct. Effects of sympathetic and parasympathetic nerves on bile duct motility have not been demonstrated. In the present study, therefore, we examined vagal and sympathetic effects on bile duct motility in the pigeon.

Pigeons were anesthetized by intramuscular administration of urethane (0.8 g/kg, Nacalai Tesque, Kyoto, Japan). A tracheal cannula was inserted, and artificial ventilation was continued throughout the experiments. After a midline incision, the abdominal cavity was opened widely. The left bile duct was exposed. Using a bipolar needle electrode, as shown in previous reports (2, 5), an action potential consisting of two to five spikes synchronous with peristalsis was recorded from the outer muscle layers of the duct. Effects of nerve stimulation were qualitatively assessed by changes in the frequency of action potentials (peristalses), since spontaneous peristalses occurred periodically with very long and irregular intervals, as reported by Tateya (5).

* To whom correspondence should be addressed.

Peripheral cut end of the right cervical vagus nerve was stimulated with a bipolar platinum electrode (10–20 Hz, 2 msec, 3–5 V) for 1 to 5 min at appropriate intervals, usually every 15 min. Vagus nerve stimulation increased the frequency of peristalses (Fig. 1). The excitatory effect on bile duct motility produced by the vagus nerve stimulation was abolished by intravenous administration of atropine sulfate (0.2–1 mg/kg, Merck Co., Darmstadt, West Germany), a muscarinic receptor antagonist, and hexamethonium bromide (2 mg/kg, Sigma Chem. Co., St. Louis, MO, USA), a nicotinic receptor antagonist (Fig. 1A, B). Tetrodotoxin (5–10 μg/kg, i.v., Sankyo Co., Ltd., Tokyo, Japan), an agent that blocks action potentials in nerves, abolished the increase in the frequency of peristalses by vagus nerve stimulation (Fig. 1C). Therefore, vagal cholinergic preganglionic efferents connecting with postganglionic cholinergic neurons may be considered to cause excitation of motility of the bile duct via muscarinic receptors.

As shown in Fig. 1D, peristalses occurring spontaneously after administration of atropine (0.3 mg/kg, i.v.) were ceased by vagus nerve stimulation in some pigeons. This inhibitory effect was abolished by tetrodotoxin (10 μg/kg, i.v.). The bile duct may also be innervated by nonadrenergic-noncholinergic inhibitory neurons whose existence in the vagal innervation is well-known in the mammalian gastrointestinal tract.

To examine the effect of sympathetic efferents on bile duct motility, spinal segments numbers 16 and 21, corresponding to the thoracic segments of mammals, were transected after laminectomy. Two platinum plate electrodes were placed on the surface of the spinal cord at the rostral and caudal ends of the separated segment, respectively. The segment to be stimulated was shielded from the surrounding tissues by liquid paraffin. Electrical stimulation of the spinal cord (5–20 Hz, 1 msec, 3–5 V) for 2 min at 10-min intervals inhibited spontaneous peristalses of the bile duct (Fig. 2A). After administration of propranolol hydrochloride

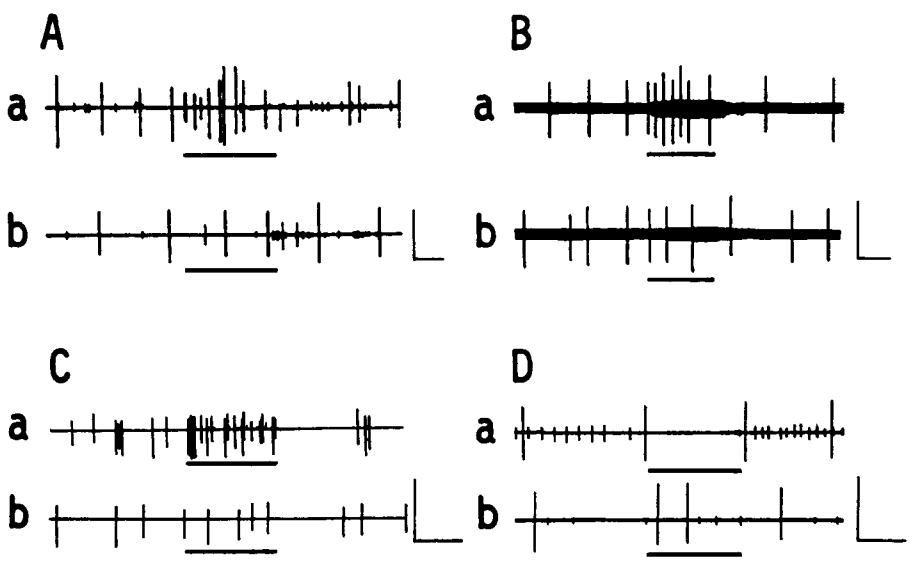

Fig. 1 Effects of intravenous administration of atropine (A, 0.2 mg/kg), hexamethonium (B, 2 mg/kg) and tetrodotoxin (C, 10 μg/kg) on the excitatory response of action potentials (peristalses) to vagus nerve stimulation, and the effect of tetrodotoxin (D, 10 μg/kg) on the inhibitory response to vagus nerve stimulation after administration of atropine (0.3 mg/kg). Bars below each trace show vagus stimulation (10 Hz, 2 msec, 5 V). A to D were obtained in 4 different animals: a in A to C, control responses; a in D, response 15 min after administration of atropine; b in A to D, responeses 15, 10, 8 and 10 min after administration of drugs, respectively. Vertical calibrations: 0.5 mV in B, 1 mV in A and D, and 4 mV in C. Horizontal calibrations: 1 min in A–D.

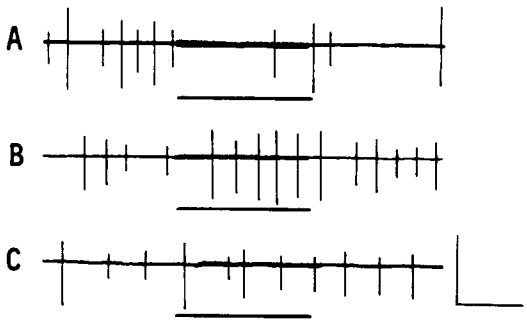

Fig. 2 Effects of subcutaneous administration of propranolol (B, 30 mg/kg) and successive adminstration of phentolamine (C, 30 mg/kg) on the response of action potentials (peristalses) to stimulation of the sympathetic area in the spinal cord (20 Hz, 1 msec, 5 V). Periods of stimulation are shown by underlining. A, control; B, response 15 min after administration of propranolol; C, response 50 and 20 min after administration of propranolol and phentolamine. Calibrations: 0.5 mV and 1 min.

(30 mg/kg, s.c., ICI Ltd., London, UK), a β-adrenoceptor blocking agent, spinal cord stimulation elicited an increase in the frequency of peristalses (Fig. 2B). This excitatory effect was abolished by successive administration of phentolamine methanesulfonate (30 mg/kg, s.c., Ciba Geigy Ltd., Basel, Switzerland), an α-adrenoceptor blocking agent (Fig. 2C). Administration of propranolol and phentolamine in doses lower than 30 mg/kg, s.c. had no considerable effects. The results revealed that sympathetic nerves inhibited bile duct peristalses via β-adrenoceptors and excited peristalses via α-adrenoceptors.

In the pigeon, vagus nerves act as accelerators and sympathetic nerves act as attenuators of bile duct motility. Such features of the innervation to the bile duct in the pigeon are similar to those to the sphincter of Oddi or the sphincterial area in mammalian species such as the dog, cat and guinea pig (6-9).

References

1. Boyden EA: The comparative anatomy of the sphincter of Oddi in mammals, with special reference to the choledochoduodenal junction in man; in Biliary System, Tayler ed, Blackwell, Oxford (1965) pp 15-40.
2. Yamasato T: Physiological and pharmacological studies on the motility of the bile duct in the chicken. Jpn J Smooth Muscle Res (1974) **10**, 287-297 (in Japanese).
3. Ryan JP: Motility of the gallbladder and biliary tract; in Physiology of the Gastrointestinal Tract, Johnson ed, 2nd Ed, Vol 1, Raven, New York (1987) pp 695-721.
4. Schmidt CR and Ivy AC: The general function of the gallbladder. J Cell Comp Physiol (1937) **10**, 365-383.
5. Tateya K: The motility of the bile ducts in pigeons and the effects of some autonomic drugs on it. Jpn J Smooth Muscle Res (1979) **15**, 295-308 (in Japanese).
6. Crema A, Berte F, Benzi G and Frigo GM: The responses of the sphincterial area of the extrahepatic biliary tract to the stimulation of sympathetic and parasympathetic nerves. Acta Physiol Latinoam (1964) **14**, 24-32.
7. Nakayama S: Nervous and chemical regulation of the movements of biliary tract. Okayama Igakkai Zasshi (1969) **81**, 1-11 (in Japanese).
8. Persson CGA: Dual effects on the sphincter of Oddi and gallbladder induced by stimulation of the right great splanchnic nerves. Acta Physiol Scand (1973) **87**, 334-343.
9. Nakayama S and Fukuda H: Conduction of activity between muscles in the terminal region of the common bile duct and in the neighbouring duodenum. Acta Med Okayama (1976) **30**, 21-35.

Received January 17, 1990; accepted March 12, 1990.