Effects on A–V Conduction of Tetrodotoxin Selectively Injected into the A–V Node Artery of the Dog

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IIJIMA, T., MOTOMURA, S., Taira, N. and HASHIMOTO, K. Effects on A-V Conduction of Tetrodotoxin Selectively Injected into the A-V Node Artery of the Dog. Tohoku J. exp. Med., 1972, 107 (1), 99-100 — Tetrodotoxin was injected selectively into the A-V node artery of the dog heart in situ, of which both vagi and both cardiac nerves were cut. Tetrodotoxin at doses of 1 to 3 µg abolished effects of electrical stimulation of either the left vagus or the left cardiac nerve on A-V conduction without any direct effect on A-V conduction. A very large dose of tetrodotoxin decelerated A-V conduction. These results led to the conclusion that cells concerned with major part of the A-V delay was less affected by tetrodotoxin than the autonomic nerve fibers that cause decisive effect on A-V conduction. ——— tetrodotoxin; A-V conduction; autonomic nervous system

It has been reported that tetrodotoxin given into the S-A node artery blocks only the transmitter release by nerve excitation at the S-A node (Tomlinson and James 1968, Hashimoto and Chiba 1969). Chiba et al. (1969) described that injection of tetrodotoxin into the A-V node artery caused A-V block. Present experiments were performed to get further information about the effect of tetrodotoxin on A-V conduction.

Experiments were performed on 6 dogs anesthetized with sodium pentobarbital. After heparinization the direct perfusion of the A-V node artery was performed which was originally devised by Nadeau and Amir-Jahed (1965). The vagi and the cardiac nerves were cut bilaterally. After the S-A node was destroyed, the right atrium was driven at intervals of 400 msec. Bipolar recording electrodes were attached to the right atrial and the left ventricular epicardium. The A-V conduction time was measured with an “A-V interval graph” (Taira et al. 1971). The distal end of the cut accelerator branch of the left stellate ganglion and that of the left vagosympathetic trunk were stimulated with pulses of 0.1 msec duration and varied voltage at 10 Hz.

When injected into the A-V node artery, tetrodotoxin at doses of 1 to 3 µg abolished both maximum positive dromotropic effect of the left cardiac nerve stimulation (Fig. 1) and maximum negative dromotropic effect of the left vagosympathetic trunk stimulation. There were no effects by increasing stimulus parameters (duration, voltage and frequency) of the nerves. Either A-V conduction or dromotropic effect of l-norepinephrine was never changed at that dosage of tetrodotoxin. Further increase in doses of tetrodotoxin caused negative dromotropic effect on A-V conduction.

These results indicate that tetrodotoxin blocks the effect of nerve stimulation at one-third threshold dose to decelerate A-V conduction.

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Fig. 1. Effect of tetrodotoxin on positive dromotropic responses to left cardiac nerve stimulation. S: artifacts of atrial stimulation. A and V: time of atrial and ventricular excitation. A-V intervals indicate the A-V conduction time. NS: left cardiac nerve stimulation (0.1 msec, 12 V, 10 Hz and for 30 sec). NS': left cardiac nerve stimulation (0.2 msec, 24 V, 30 Hz and for 30 sec).

References


